THE
LARYNGOSCOPE.

VOL. XXVIII. ST. LOUIS, MAY, 1918. No. 5.

ORIGINAL COMMUNICATIONS.

(Original Communications are received with the understanding that they are contributed exclusively to THE LARYNGOSCOPE.)

CARTILAGINOUS TUMORS OF THE LARYNX.*

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Cartilaginous tumors of the larynx occur but rarely. Only 38 cases have been reported in the literature. In 1900 Alexander reported 29 cases tabulated since 1834, and in 1909 Mansfeld reported 9 cases. In addition, I have been able to collect 8 cases from the literature, and one of my own, making 47 in all. In three of Alexander's cases, there was general hypertrophy of one or more cartilages of the larynx; it seems questionable therefore, whether these cases should be classified with true laryngeal tumors.

The first case report was published by Froriep in 1834. In this case a large cartilaginous tumor on the left side of the larynx, a smaller tumor below the cords on the right side, and a third on the anterior surface of the posterior part of the cricoid cartilage, were found at postmortem. After the introduction of the use of the laryngeal mirror in 1859, many laryngeal tumors, previously considered rare, were noticed, and the first clinical case was reported by Türck in 1863. The patient died the day following the examination and at necropsy a tumor of the cricoid cartilage was found. In 1888, Bruns collected 10,747 case reports of laryngeal tumors and found only 14 cases of cartilaginous tumors. The first work of importance on cartilaginous tumors of the larynx was not

*Presented as a candidate's thesis to the American Laryngological, Rhinological and Otological Society, 1917.

brought out until 1900, when Alexander's very thorough study on the subject was published.

Virchow classified such tumors as: (1) *Ecchondromas,* that is, cartilaginous tumors that are an outgrowth of the mother substance, like the branch of a tree, and do not invade the original tissue, and (2) *enchondromas* which develop from a non-cartilaginous matrix. If this classification is accepted, all cartilaginous tumors of the larynx would be classified as ecchondromas, as they all originate in pre-existing cartilage. Alexander accepted the term ecchondroma as defined by Virchow and added four other groups of tumors: (1) *Chondromas,* true neoplasms invading the mother substance from which they originated and which in most cases are malignant; (2) *mixed tumors,* tumors made up of different tissues, i. e., fibro-enchondromas, myxochondromas, chondrosarcomas, and chondromyxomas; (3) *inflammatory*—in this group but one case of questionable etiology was reported, and (4) *general hypertrophy* of one or all the cartilages of the larynx, of which three cases are noted in Alexander's statistics but none since.

It would seem that in the classification of cartilaginous tumors of the larynx, the terminology should designate whether the tumor is benign or malignant. A chondromyxoma may be benign or malignant, but the name would not indicate this fact. A terminology indicating the combined microscopic and clinical conditions would be ideal but until there is such a classification, I shall accept that given by Alexander. The following is the report of a case of chondroma of the thyroid and cricoid cartilages.

P. E. O., 140068, male, aged 44 years, a farmer, was examined Sept. 1, 1915. He had had diphtheria thirty years previously and typhoid twenty-two years previously. There was no history of specific disease. He complained chiefly of having been hoarse for four years. When twenty-one years of age (twenty-three years previously) he was hit by a baseball on the right side of the neck, over the larynx. For a short time talking pained him, but he was never hoarse. Following a cold he noticed a lump on the outer side of his neck. This was never sore nor tender. One year previous to the present examination he consulted a physician, but nothing was done until six months later, when his home physician performed a tracheotomy on account of an attack of dyspnea. No other treatment was given. There was no loss of weight; no dysphagia. Although his voice was husky, he could make himself understood by plugging the tracheotomy tube with his finger.

Examination.—In the external examination of the neck a tumor was found 1.5 by 1.5 inches in size and extending to the right from the midline; its upper limit was one inch below the upper margin of the thyroid cartilage. It was rounded in outline, bony, hard, and fixed to the side of the larynx; the skin covering it was normal. The tracheotomy tube was in place. In examining the larynx the right cord, the false cord and aryteno-epiglottic fold were found displaced toward the midline, a small slit being left for the passage of air. The tumor could be seen bulging on the right side of the larynx and covered by mucous membrane normal in appearance.

Fig. 1. Chondroma of the larynx. Right side of the midline of neck.

The roentgen and general examinations were negative. The examination other than the laryngeal findings was negative. *Diagnosis*: Cartilaginous tumor of the larynx (Figs. 1 and 2).

Sept. 4, 1914, an operation was performed under novocain anesthesia (E. S. J.). An incision to the right of the midline exposed the rounded hard tumor which was found to have invaded the thyroid and cricoid cartilages and the upper part of the trachea. Immediate microscopic examination of the tissue (frozen section) showed the growth to be a chondroma (Fig. 3). The tumor could have been shelled out, but as this would have necessitated a large opening into the larynx and trachea which would destroy the voice, it was thought best to leave a thin shell of the tumor and remove

entirely the outer part, the more radical operation to be performed later should the condition recur. The wound was closed primarily with a small pack in the lower portion. The convalescence was favorable and the patient was discharged about two weeks after the operation. When he returned for examination April 6, 1916 there had been no change in the voice and no sign of the return of the tumor. Externally, the neck was normal except for the scar and the tracheotomy tube. The larynx was in the same condition as before the operation except that there was more room for the passage of air.

Etiology. Nothing definite seems to be known regarding the etiology of these cartilaginous tumors. Several hypotheses have

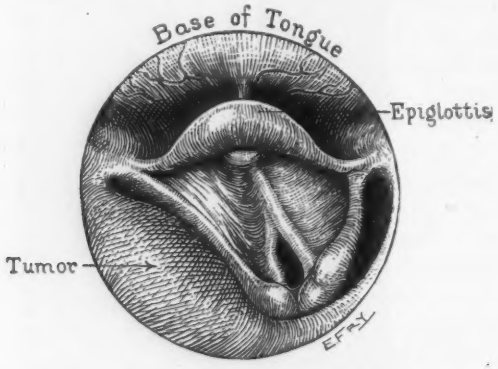

Fig. 2. Displacement of the right side of larynx. Due to chondroma of the thyroid cartilage.

been advanced, but none is borne out by the facts. Bertoye has suggested that the tumors are in some way associated with ossification of the cartilage of the larynx which begins during the fifth decade. Alexander in his tabulation cites the cases of 7 patients under 40 years of age; Mansfeld cites 3 cases, and I have been able to collect 2, making 12 of a total of 47 in which the tumor was present in patients under 40 years of age. This would not seem to substantiate Bertoye's theory. Eppinger thought that possibly the cartilage proliferation occuring about the joints at times might be analogous to cartilaginous tumors, but the fact that the tumors appear on the solid portion of the larynx, away from the moveable parts, does not verify this assumption. The question of chronic inflammation as a factor has been considered, but there are not sufficient data to establish such etiology.

My patient gave a history of injury of the side of the larynx by a blow from a ball 19 years before the onset of hoarseness, but whether or not the injury was a factor in the etiology of the tumor is not known. However, this case seems to be the only one in the literature in which the etiologic factor of trauma might be considered. It is probable that until we discover the cause of tumors in general we shall not determine the etiology of cartilaginous tumors of the larynx.

Symptomatology. More than half of the cases found in the literature occurred in the fifth and sixth decades, although one case was reported of a girl 17 years of age, and 5 cases that occurred in the seventh decade. Most of the patients were men; only 6 women have been noted. The symptomatology of these tumors is due to interference with the function of the larynx, either respiratory or vocal, and interference with swallowing. The symptoms are variable and depend on the location of the tumors, their size and rate of growth. Hoarseness is usually an early symptom, but if the tumor is on the epiglottis or the arytenoid and does not interfere with the cords, this symptom will not be noticed. The same is true when the tumor is on the cricoid and entirely subglottic. Roos, in 1910, reported a case of subglottic tumor in which there were marked symptoms of dyspnea for five months, but no hoarseness. In the case of a tumor of the thyroid, hoarseness is usually an early symptom unless the tumor extends externally, in which case laryngeal symptoms are noted late.

Respiratory difficulty is dependent on the size of the tumor and the rate of the growth. Very large tumors may be present in the larynx, and the breathing space may be surprisingly small, but on account of the slowness of growth the symptoms of respiratory obstructions are not marked. A rapidly growing tumor causes pronounced symptoms. Cricoid tumors that usually spring from the posterior surface of the anterior part of the cartilage cause respiratory difficulties early because of their location. Tumors involving the cricoid, as in Bolms' case and Moure's second case, may cause difficulty in swallowing if they extend toward the esophagus. Boerger's patient (Mansfeld's group) swallowed with difficulty for five years and was hoarse for a shorter period. Bolms' patient gave a history of an external tumor of the neck for one year, hoarseness for six months, and dysphagia for three months. With the larger tumors there is a sense of fullness and pressure and in some instances the patient makes a heaving noise described as resembling the inspiratory spasm of whooping cough. Three

patients (those of Froriep, Türck, and Pretelle, reported by Alexander, died of dyspnea. Many patients have had to have emergency tracheotomies.

Location of tumors. In my group of nine cases (eight from the literature and one personal) the cricoid was involved in eight, the thyroid in two, and the arytenoid in one. As in Moure's and Daure's patient and in mine, the thyroid and cricoid were both involved; these patients are included in the eight cases of the cricoid group and the two cases of the thyroid group. In one of Garel's two cases the tumor was probably an ecchondroma, but as this is questionable, I have designated it as indeterminate. The following table shows the cartilage involved in my group of cases:

TABLE 1.

Cricoid 8	
Ecchondroma	1
Chondromas	5
Indeterminate	1
Mixed	1
Thyroid 2	
Chondromas	2
Arytenoid 1	
Ecchondromas	1

The cartilages involved by the different types of tumors in Alexander's, Mansfeld's and the writer's cases were as follows:

TABLE 2.

Cricoid 24	
Ecchondromas	7
Chondromas	11
Mixed tumors	5
Indeterminate	1
Thyroid 14	
Ecchondromas	5
Chondromas	7
Mixed tumors	2
Arytenoid 4	
Ecchondromas	4
Epiglottis 3	
Ecchondromas	3

It will be seen from the table that the arytenoids and epiglottis are attacked by ecchondromas only, while the cricoid and thyroid cartilages may be involved by any of the types of tumors. The cricoid is involved nearly twice as frequently as the thyroid. Chondromas or mixed tumors do not involve the arytenoids or the epiglottis.

Objective findings. The normal appearance of the mucous membrane, not alone surrounding the tumor, but in the entire larynx, and the absence of signs of congestion, unless some temporary acute in-

fection is present, are characteristic in cases of cartilaginous tumors. The hardness felt when touching the tumor with a probe is different from that of any other type of laryngeal tumor, although in some cases, as in the case reported by Roos, the tumor is somewhat soft. Cartilaginous tumors of the larynx arise from one of the cartilages of the larynx, so that the location of a tumor may be of value also in differentiating it from other types. An external tumor of the neck fixed to the larynx, very hard without signs of inflammation, would suggest a cartilagious tumor. In some cases the roentgen ray shows

Fig. 3. Cartilaginous tumor of the larynx (photomicrograph).

a shadow which is of value in making the diagnosis. The question of malignancy in these cases is usually ruled out without difficulty but must be considered. The absence of glandular enlargement is a characteristic feature although this point in itself is of little value, since other benign tumors have no glandular enlargement and even epitheliomas may exist for years without metastasis. Fränkel in 1893 reported a case in which six months following a radical operation for chondroma of the cartilage, two glandular swellings developed in the neck. These were removed and on examination proved to be "slimy, softened chondromatous glands." This is the only case reported in which a cartilaginous tumor of the larynx developed metastasis.

Cartilaginous tumors of the larynx vary in size from that of a small tumor on the arytenoid to that of a tumor involving the thyroid and cricoid cartilages and also the upper rings of the trachea. The ecchondromas are usually the small tumors; the chondromas and mixed tumors, as a rule, grow to a much larger size. The ecchondromas grow slowly while the chondromas are usually more rapid in development. The only safe way to make an absolute diagnosis is to remove tissue for microscopic examination.

Histopathology. Detailed descriptions of the microscopic pathology of cartilaginous tumors of the larynx are not frequent in the literature. The ecchondromas are the same in structure as the cartilage of the larynx, consisting of hyaline cartilage. Sometimes ossification is seen.

In referring to chondromas, Alexander states that there is an irregularity in the size, shape and arrangement of the cells, and that the cartilaginous tissue shows an unusual energy of growth. In the older tumors, cyst formation may occur from a retrogressive change in the tumor. Calcification may also be seen. The mixed tumors present pictures too varied to permit a review of their microscopic appearance. In regard to the histology of inflammatory tumors, and the general hypertrophy group, nothing is noted in the literature.

Treatment and prognosis. Small ecchondromas may be removed by the endoscope and this seems the method of choice although in some instances it is necessary to do a thyrotomy. The chondroma and mixed tumors present a more difficult problem. In almost all cases of the latter there is some dyspnea. Three patients of Alexander's group died of suffocation before tracheotomy could be done. In considering the operative treatment of these patients, it must be remembered that the voice should be preserved as long as possible. If the tumor is not malignant and its removal would destroy the voice, some conservative treatment is indicated. The end results of cases in which a radical operation has been done further impresses this feature. However, if the tumor is malignant, the most radical operation should be done.

End results in 22 cases of chondromas and mixed tumors (Alexander's, Mansfeld's and the writer's statistics). Three patients choked to death without tracheotomy; 1 died following tracheotomy. Fifteen had external operations varying from complete laryngectomy to thyrotomy and partial or complete removal of the tumor. One patient had roentgen treatment and is reported "better": 1

Case No.	Author	Sex	Age	Subjective History	Objec
1	GAREL, J.:	F	35	Patient came for throat trouble, vocal and respiratory.	Below the left with its con midline of t was hard an
2	DURAND AND GAREL, J.: Les chondromes du cartilage cricoide. *Ann. d. mal. de l'oreille, du larynx*, 1908, XXXIV 629-654.	M	57	Ten months' history. Began by dyspnea on exertion, increasing in intensity and frequency. Voice not a good tone. Breathing on exertion resembled the respiratory spasm of whooping cough.	Laryngeal ex round, regul subglottic r vocal cord, mucous mem operation at ted to it 7 m nea became
3	LANDWEHRMANN: Enchondrose am Giessbeckenknorpel. *Ztschr. f. Ohrenh.* 1909, LIX, 380.	F	28	Hoarseness for 2 years.	Nodular grow process, und right true co millimeters During phon opposite side
4	MOURE, E. J. AND DAURE: Enchondrome volumineux du larynx. *Jour. de Med. de Bordeaux*, 1910, XL, 43.	M	52	Six years ago noticed difficulty in speaking and weakening of normal timbre of his voice. Laryngeal examination showed a tumor the size of a nut on left side of larynx. Functional disturbances continued more marked. Hoarseness and dysphagia for 2.5 years. Dyspnea at times for 2 years. General health good.	Tumor, hard, the external extending d displacing Gland in car hard, movab tion showed aryteno-epig round tumor orange and of the lary membrane r vessels. R showed a s the thyroid ceeded the and extende
5	MOURE, E. J.: Des enchondromes du larynx. *Rev. hebd. de Laryngol*, 1910, *XXX*, 48-61.	M	56	For one year increased respiratory difficulty which was thought to be cardiopulmonary. For 2.5 weeks condition had been worse. One severe attack of dyspnea.	Marked dyspn subglottic r tion, multi seen mostly trachea; no
6	ROOS: Myxochondroma laryngis subglotticum. *Ztschr. f. Ohrenh.*, 1910, LXII, 228-30.	M	44	Dyspnea on slight exertion noticed 4 or 5 months before; became progressively worse. Much trouble when lying down. No cough, no dysphagia, no hoarseness.	Slight respir more mark examinatio nothing pa sinuses. clear. Lary in function the cord or right wall membrane. and to the
7	BOLMS, J.: Über zwei Fälle von Chondrom am Ringknorpel. Bonn, Menzel, 1912, 32 p.	M	66	One year before, following influenza, patient noticed swelling of the throat. A tumor enlarged slowly until the size of a fist. Six months later hoarseness developed. Some dysphagia for 3 mos. When examined some dyspnea on exertion. No weight loss.	Firm, nodula circumscri sels and limits of l largement. to right. cord. Muc normal. tion visibl a bowing
8	SALOMONSEN, K.: (Clinical contribution on enchondro-	M	56	Six months' hoarseness and dyspnea on exertion. No	Laryngeal glottis ask

TABLE 3.

ry	Objective Findings.	Operation	Location of Tu
throat respira-	Below the left vocal cord was a tumor with its convexity extending to the midline of the trachea. The tumor was hard and resisted the probe.	Thyrotomy. Tumor operated upon.	Cricoid cartilage
. Be- xertion, lity and not a ing on the res- whoop-	Laryngeal examination showed a round, regular, hard tumor in the subglottic region under the left vocal cord, covered by a normal mucous membrane. Patient refused operation at this time but submitted to it 7 months later, when dyspnea became marked.	Low tracheotomy was done; operation 1 month later. Incision extended through the thyroid cricoid and first 2 rings of trachea Tumor the size of a nut was seen on the posterior wall of cricoid. The tumor was curetted off and the wound closed.	Cricoid.
rs.	Nodular growth on the right vocal process, under the origin of the right true cord. Firm growth a few millimeters in length and width. During phonation it moved to the opposite side.	Removed with double curet.	Arytenoid.
d diffi- d weak- nbre of eal ex- tumor on left nctional ntinued rseness or 2.5 times health	Tumor, hard, globular and fixed on the external left side of the larynx, extending down to the trachea and displacing larynx to the right. Gland in carotid region on left side; hard, movable. Laryngeal examination showed on the level of left aryteno-epiglottic fold a smooth round tumor the size of a mandarin orange and concealing a large part of the laryngeal cavity. Mucous membrane normal except for dilated vessels. Radioscopic examination showed a shadow in the region of the thyroid cartilage which exceeded the limits of the cartilage and extended down over the trachea.	Preliminary tracheotomy under cocain. Three weeks later operation under cocain. A thyrotomy was done in the midline, exposing a multi-lobular round tumor which had invaded the inferior part of the larynx and also the pyriform sinus. The neoplasm was adherent to the posterior surface of the cricoid cartilage.	Thyroid and cri
ed res- which cardio- weeks worse. f dysp-	Marked dyspnea. Voice husky. In subglottic region, on deep inspiration, multilobular tumor could be seen mostly on the right side of the trachea; not palpable externally.	Tracheotomy under cocain. Operation under cocain. Upper 5 rings of trachea incised. The tumor mostly on left side bulging the trachea and forming a diverticulum, was removed in pieces and by curet. Growth the size of a chestnut.	Thyroid, cricoi trachea.
xertion ths be- ssively e when igh, no ness.	Slight respiratory stridor at rest; more marked on exertion. General examination negative. Externally nothing palpable in the neck. No sinuses. Throat negative. Voice clear. Larynx normal. Cord normal in function. Tumor visible below the cord on posterior portion of the right wall covered by normal mucous membrane. Small slit posteriorly and to the left for passage of air.	Ether-morphin anesthesia Midline incision. No previous tracheotomy. Upper rings of trachea, cricoid and lower half of thyroid cartilage divided. A soft tumor the size of a cherry arising from upper and posterior part of cricoid cartilage exposed. Its base was size of a coffee bean. The tumor was excised and curetted, the base cauterized, the wound closed primarily.	Cricoid.
ing in- noticed at. A vly un- t. Six ess de- phagia amined ertion.	Firm, nodular, fixed tumor, definitely circumscribed over left jugular vessels and extending above upper limits of larynx. No glandular enlargement. Displacement of larynx to right. Immobility of left vocal cord. Mucous membrane of larynx normal. No localized tumor formation visible. Roentgen ray showed a bowing of trachea to the right.	Operation under ether. Curved incision over highest portion of the tumor. Tumor fixed to cricoid cartilage; also to trachea but not to thyroid, was removed. Tumor had entirely destroyed portion of cricoid cartilage, and upper 3 rings of trachea had been involved.	Cricoid cartilage
ss and	Laryngeal examination shows the	Ether anesthesia. Low trach-	Cricoid cartilage

ation of Tumor.	Pathologic diagnosis	End Results
id cartilage.	Enchondroma.	Several days after operation pulmonary complications caused death.
id.	Enchondroma.	No recurrence.
noid.	Cartilaginous tumor.	Eight weeks after removal of tumor, voice not clear because of partial adduction paralysis.
oid and cricoid.	Benign chondroma.	Not stated.
oid, cricoid and chea.	Fibrochondroma.	Acute myocarditis one week after operation. Died. Necropsy: Tumor much more extensive than supposed. Cricoid cartilage entirely deformed and friable. Lumen of cricoid small. Tumor extended into submucous layer of esophagus and down to 6th ring of trachea. Operation removed portion of tumor that projected into trachea.
oid.	Hemmorrhagic myxochondroma (benign).	7¼ months after operation the larynx was normal. No recurrence.
oid cartilage.	Chondroma.	Not stated.
oid cartilage.	Cartilaginous tumor.	Tumor incompletely removed.

2	DURAND AND GAREL, J.: Les chondromes du cartilage cricoide. *Ann. d. mal. de l'oreille, du larynx*, 1908, XXXIV 629-654.	M	57	Ten months' history. Began by dyspnea on exertion, increasing in intensity and frequency. Voice not a good tone. Breathing on exertion resembled the respiratory spasm of whooping cough.	Laryngeal exan round, regular, subglottic reg vocal cord, co mucous membr operation at t ted to it 7 mon nea became ma
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8	SALOMONSEN, K.: (Clinical contribution on enchondroma of the larynx). *Hosp. Tid.* 1914, 5 R. VII, 963-972.	M	56	Six months' hoarseness and dyspnea on exertion. No dysphagia. Hoarseness and dysphagia increasing.	Laryngeal exan glottis askew f front to the le the subglottic r right vocal co growth the si a broad base. normal. No ul

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ss and n. No ss and	Laryngeal examination shows the glottis askew from the right side in front to the left side posteriorly. In the subglottic region underneath the right vocal cord was seen a round growth the size of a cherry with a broad base. The vocal cords were normal. No ulceration.	Ether anesthesia. Low tracheotomy incision extended up through the trachea and cartilage. A tumor the size of a walnut visible in right subglottic region curetted out. Wound completely closed.	Cricoid cartil

icoid.	Enchondroma.	No recurrence.
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ricoid cartilage.	Cartilaginous tumor.	Tumor incompletely removed.

had repeated endolaryngeal operations with "good results"; 1 was not operated on and the subsequent history is not known. Of the 15 patients operated on, 5 died from pulmonary complications shortly after operation and 5 improved for varying periods of time, the length of which was not stated. One patient was doing well seven and one-half months after operation; the condition of 2 others was good after the operation but the end results are not stated. In 1 the condition "relapsed" and in the case of another the tumor was incompletely removed and the results are not stated. (Table 3).

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SOME OBSERVATIONS ON THE DECOMPRESSION OPERATION ON THE HYPOPHYSIS BY THE NASAL ROUTE.*

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In working in any comparatively new field of surgical endeavor various problems arise that with further experience become simpler in their performance and less complicating in their results.

The operation for the decompression of the pituitary body may be carried out by several different routes. The transtemporal, the transfrontal and the transphenoidal being the usual ones followed. The first two are not considered in this paper. The transphenoidal route has been used in several different ways, namely, through the mouth, through the antrum Highmore and through the nose. The nasal route as originally carried out was a most serious and radical performance. The earliest operators, like Schloffer, Eiselberg, Proust and Ollier, commenced with an external excision about the nose, including the turbinates, septum and ethmoid cells. Later, was followed by the removal of most of the structures within the nose, including the turbinates, septu mand ethmoid cells. Later, particularly following the work of Oscar Hirsch, this sacrifice of important structures was found to be not only unnecessary but also harmful. The technique as now employed through the nose gives ample room for working with the minimum of tissue loss, and to the rhinologist in particular the operation offers an exceptionally favorable avenue by which to approach the floor of the sella turcica. If he is skilled in the performance of submucous septal resection, he can with facility and rapidity open widely into the sphenoid cavity, which then gives him easy access to the sella floor and the gland that lies within. With his perfected knowledge of the anatomy of this region and of the variations and anomalies that may be expected, the rhinologist is particularly fitted for this operation.

The general surgeon when following the nasophenoidal route usually commences his incision external to the nose. Kanavel incises the upper lip around the alae. Cushing incises beneath the lip at the labiogingival junction. In following the antrosphenoidal route the incision is made at the labiogingival junction. Both Citelli and West

*Read at the sixty-seventh annual meeting of the Illinois State Medical Society, Bloomington, Ill., May 8, 1917.

commence their resection of the septum further back and do not attempt a submucous procedure.

The criticism to these various methods is the greater likelihood of infection following operation, besides the necessity of removal of considerable tissue, much of which is functionally important and the removal of which later produces distressing symptoms. In many of the operations as carried out by the general surgeon, systemic anesthesia is used, which adds seriously to the case and is unnecessary. I believe the ideal anesthesia to be morphin-hyoscin or scopolamin-morphin with flake cocain locally on the septum and sphenoid. I would offer a suggestion and sound a note of warning regarding the preparation for anesthesia. A careful and reliable examination of the urine for evidences of oxybutyric and diacetic acids and acetone should be made in all cases to avoid the possibility of acidosis developing after operation. A reasonable food consumption should precede the operation for dieting or fasting may develop this condition. Also hurried operations should be deprecated. The only case I lost from operation of this kind was of this nature. In brief it was as follows:

A man, 47 years of age, having only central vision, blindness commencing two years before, both fundi showing beginning peripheral atrophy; headaches; no perverted pituitary symptoms. Owing to certain circumstances no routine physical examination including urinalysis was made in the hospital. He was operated by request without any previous observation by me aside from a preliminary nasal inspection. Scopolamin-morphin and cocain anesthesia. The patient dozed throughout the operation. On entering right sphenoid encountered a cyst which did not rupture until I entered the left sphenoid. Two drams or more of straw colored fluid, slightly blood stained, escaped. All of the floor of sella was absorbed from pressure by the cyst. The operation was the simplest, quickest and easiest of my operations. No tissue was removed. The patient had a restless night, vomited constantly, severe headache, temperature at first 97 8-10, pulse 80, later temperature rose to 102 8-10. A condition of acidosis was diagnosed from the symptoms of vomiting, pulse, headache, urine examination and breath. Exitus lethalis occurred on the second day following operation.

Ordinarily a diagnosis of these cases is established by a triad of symptoms, namely, headache, involvement of vision and perverted physiologic pituitary function, plus the Roentgenographic findings. The Roentgenogram if correctly taken and understandingly read is valuable both to the operator and the diagnostician, but especially

to the operator, because it furnishes him with evidence of capacity and dimensions. A good picture will show the size of the sphenoidal cavities, a widening and deeping of the sellar floor, even a shadow outline of the glandular mass if it has already invaded the sinus, a thickening or a thinning of the bones at the base of the skull; enlargement or absorption of the clinoid processes and an increase or decrease of the diaphragm opening. This knowledge is of supreme importance to the surgeon because of the variations met with in the size, shape and position of the sphenoid sinus, as well as in the thickness of its walls and the relationship of adjacent parts, like the posterior ethmoid cells, the optic nerves and the carotid arteries.

In any series of normal head sections some divergence will be found in the anatomy of this keystone bone. Ordinarily the floor of the sella presents at about the superior posterior angle of the inner wall of the sphenoid cavity. A distinct promontory can be seen in the center, indicating the most dependent part of the pituitary fossae. The bone at this point is usually quite thin and easily broken through. Many of the descriptions of this operation do not describe this part of the operation with sufficient clearness or detail. The promontory indicating the floor of the sella may be absent and in place of a convexity either a straight or even a concave wall may present itself. Now if you were to follow the directions given by some authors to enter through the roof or by that of others to perforate the posterior wall you would in some instance enter the anterior cerebral fossae or the middle fossae, jeopardizing the chiasm and the cavernous sinus. Occasionally one finds a hard and thick wall in the region of the promontory. This makes progress slow and, besides, adds uncertainty to the work. In one case of cadaver operation I found a large posterior ethmoid cell interposed between the sphenoid cavity and the sella, which of course had to be broken through first before one could enter the fossae. Onodi in his work on sinuses has shown several variations of this kind. But whatever anatomic conditions may be present it is always necessary for the operator to keep working in the median line to escape injury to nerve, artery and cavernous sinus. This in itself is a strong argument in favor of the septal route. You are always working in the median line from the beginning and that spells safety first. One of the advantages of working submucously is that the entire operation is confined between two muco-periosteal flaps and with proper aseptic technique these flaps can be brought together at the final, thereby avoiding any danger of after infection. A case bearing out one of the variations met with was where headache

was the chief symptom and in which no eye symptoms were present. The Roentgenogram showed a depressed sella with a lengthened antro-posterior diameter. The optic chiasm most likely escaped pressure from the growth on account of its posterior position. Right here a fallacy in many books on anatomy can be exposed. That is the optic groove, which lies anterior to the pituitary fossae, is said to lodge the chiasm, which then passes from either end as the optic nerve into the optic foramen. This would give the nerve almost a right angle course, but in reality the chiasm lies further posterior, presenting in anterior border at the posterior part of the gland and the two optic nerves lying to the side. Most of the tumor cases involve the anterior lobe first and in their growth seem to meet resistance least at the floor of the fossae. The diaphragm above appears to offer greater resistance to the tumor growth than the bony floor, the bony clinoid processes before and behind and the firm dural reflections with the cavernous sinuses on the sides. One other weak point besides the floor of the fossae is the infundibular which connects the pars intermedia with the third ventricle. Along this path cysts are likely to develop. A case of this type was reported by me over two years ago. The patient made a complete recovery of all focal symptoms like headache, blindness and vertigo, after a transphenoidal decompression, and in which the gland was adenomatous and filled the sphenoid cavity. After a year, headache and vertigo began to return and four months later she died. Autopsy showed the gland greatly enlarged, filling the sphenoid sinus as well as forcing the diaphragm up in front of the chiasm. Connected with the infundibular was a cyst the size of a walnut that invaded the dilated third ventricle. This shows the two types of pathology in one case. The adenoma appeared first, later the cyst. Had the cyst been recognized it might have been drained but, as Cushing said in a letter to me about some of his cases, "Cysts even if exposed and evacuated tend to refill with return of the pre-existing visual defects." But these variations should never deter the surgeon from following this route of operative procedure, because with the knowledge of these variations and a good radio picture he can always proceed with confidence.

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THE VACCINE TREATMENT OF OZENA.*

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The history of the study of the etiology and treatment of what is called ozena is full of jubilant enthusiasm and bitter disappointments. Every now and then some investigator would joyfully exclaim: Eureka! and would describe either a new etiological cause or a method of treatment different from all hitherto in vogue. To prove that the problem of ozena has really and finally been solved the promulgation of the revolutionizing discovery would usually be accompanied by an array of overwhelming statistics of indisputable cures of ozena in an overwhelming percentage of cases, the balance being, of course, greatly or at least noticeably improved. In due time there would invariably appear a report of control investigations deducting *ad absurdum* and ridiculing the propounded theory and exposing the method of treatment as a failure.

Jonathan Wright, in two excellent articles, gave a full account of the up-to-date history of the studies of the etiology of ozena. The history of the therapeutic claims and failures, especially of the recent vaccine treatment of ozena remains, however, unwritten as yet. In the face of the triumphant statements of Perez, the prophet and of Hofer and Horn, his most ardent worshippers, a report about some investigations concerning the vaccine treatment of ozena seems almost an iconoclasm if it does not accept their etiologic theory and confirm the therapeutic wonders achieved thereby. Before, however, describing in detail our investigation of the vaccine treatment of ozena, we will give a rough historic sketch of the etiological and therapeutic theories of ozena, as the understanding of the present controversy will be thereby facilitated.

The name ozena is derived from the Greek and simply means a stench. The disease is more scientifically characterized by its other name, viz. chronic atrophic rhinitis. The affection is usually described as a chronic inflammation with subsequent atrophy of the mucous membrane of the nose, accompanied by the formation of yellowish or green crusts and by a very offensive odor. The older theories consider the atrophic condition of the nasal mucous membrane and the underlying bone as the primary factor and consequently they try to explain its origin thereby. Some of these theories, in the light of modern research, may be almost called medical superstition. Sauvager and Tillot considered congenital

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narrowness of the nasal passages, with consequent retention of moisture, as the etiological factor. Gottstein and Heymann, on the other hand, accused a too large nasal cavity as the cause of the atrophy; while Meissner holds a primitive or shallow nose with a congenital development of pavement epithelium, instead of the columnar or mucous producing variety, responsible for it. According to Zaufal a rudimentary Anlage of the inferior and middle turbinate forms the underlying condition. The so-called focal theory as expounded by Gruenwald, Hajek, Luc and in America especially by Ballenger, considers it a sclerotic atrophy due to a pre-existing inflammation of the sinuses, from where the secretions flow over the nasal mucous membrane, and eventually form dry crusts. The latter undergo decomposition and irritate the mucosa till it forms an excessive proliferation of connective tissue cells.

Some writers explain the primary atrophic condition by the extension of the blood vessels (cyanotic congestion) in a heart lesion. B. Fränkl points to the connection of ozena and the so-called atrophic catarrh, whose dried out secretions become decomposed by means of specific germs. According to Krieger, the mucous membrane undergoes dessication due to the deprivation of moisture whereby the tiny epithelial cells gradually lose their amoeboidal movements of protection and turn into the squamous type. M. Schmidt ascribes the formation of crusts to the action of dry and dusty air, especially in spring time. Cholewa and Cordes consider primary bone atrophy as the underlying cause. Freudenthal explains the origin of atrophic rhinitis by our faulty and artificial way of heating our rooms, dressing ourselves, etc., whereby a xerasia (dryness) of the interior of the nose is caused. True ozena occurs through the invasion of a thus weakened nasal cavity by a bacillus resembling the Friedlander diplococcus. This infection occurs in early life and perhaps in some cases by direct transmission from the vagina. Tuberculosis, lues and anemia have been frequently mentioned as etiological factors.

The treatment in this earlier period consisted in general medication, local applications, irrigations, electrocautery, electrolysis, operative interference upon the septum such as paraffin injection or submucous resection, opening and curetting of the sinuses, hygienic measures, etc. The different methods, highly advocated in their time, fell soon into oblivion and ozena remained an "incurable disease" till the advent of its so-called infectious theory. Among the first to describe a specific bacterium as the cause of ozena were Abel and Loewenberg. Their *bacillus mucosus capsulatus* was, however, identified with the *bacillus scleroderma* (Frisch) and declared by

Fricke to be a variety of a species of *bacillus Friedlaender*. As the only and real germs of ozena there were furthermore described the pseudo-diphtheria bacillus of Della Vedova and Belfrarts, a small, but not definitely described, organism found by Pes and Gradenigo, the bacillus fœtidus of Hajek and last, but not least, the *cocco bacillus feotidus ozenae of Perez*.

The new era of ozena investigation starts with the year 1899, when Perez published in the *Annals de L'Institut Pasteur* his researches on the bacteriology of ozena. The cocco bacillus of Perez was, however, not recognized by the medical profession and for this reason Perez (then ambassador from the Argentine Republic to Austria) asked Hofer in 1913 to prove his claim by control experiments carried out at the University of Vienna Institute for General and Experimental Pathology.

Hofer, however, did not succeed in isolating by Perez' methods the cocco bacillus from the nasal crusts of ozena patients. By injecting a portion of a bouillon mixed culture which had the characteristic fetor, into the ear vein of a rabbit, the latter developed, after several days, a purulent nasal discharge in which he found what he believed to be a pure culture of the cocco bacillus of Perez. *The important fact for us to remember is, that the cocco bacillus was not isolated from the crusts of the ozena patient but from the nasal discharge of an injected rabbit.* Hofer prepared an ozena vaccine consisting of a suspension in normal salt solution of cocco bacillus of Perez, found in the above described way and derived from seven different strains, *to which he added a culture from the nose of healthy dogs.* We will later on point to the etiological importance of the fact that the vaccine was gained from the nose of injected rabbits and contained a culture from the nose of the dog. At Chiari's Clinic, Hofer, together with Kofler, injected a number of ozena cases with this vaccine and published a report of almost marvelous cures.

Having become interested in the ozena problem by the activities of the International Committee on Ozena, I decided to investigate the claims and methods of Hofer and Kofler by working with them. Just before the outbreak of the European war, I took with Guggenheim, of St. Louis, and Horn, of San Francisco, a special course with Hofer at the Vienna Institute for General and Experimental Pathology, where we were instructed in the preparation of the vaccine and the action of the pure culture upon the injected rabbit was demonstrated to us. The turbinates of the rabbits shown to us showed only slight hemorrhages and swellings, but we were told that at a later stage purulent discharge and atrophy would follow.

The details of the vaccine preparation is fully described in the papers by Guggenheim and Horn. At Prof. Chiari's Clinic, where over a decade previous to that I had been initiated into the secrets of rhino-laryngology, an opportunity was given to us to watch the progress of the ozena patients under the vaccine treatment. Kofler, Chiari's assistant, would examine the patient's nose and throat and dictate the statements to Hofer, who would write them into the patient's history and would then inject the patient with the increasing doses of vaccine. I admired my former colleagues, Hofer and Kofler, for their optimism. After watching scrupulously for a number of weeks, I could not be convinced that even one case had been cured. As Horn, in his first paper stated correctly, we were all very pessimistic and somewhat suspicious. "Less odor, decreased number of crusts, pharyngitis turning moist," etc., etc., are very vague statements. The wish is often the father to the thought. All we American skeptics could notice was the presence of fetor, mostly of the severest type and of crusts in all noses. We took tubes of Hofer's original pure cultures of Perez bacillus with us and started to experiment with them.

Guggenheim was the first to publish a report. The calamities of the first few months of European war seem to have impressed themselves upon the mind of my friend, Guggenheim, to such a degree that his first impressions at Chiari's Clinic had become entirely dimmed. For to my great astonishment I found in his article a report of his observations during our stay in Vienna that met more than half way the optimistic and exaggerated claims of Hofer and Kofler. According to Guggenheim, of 25 cases observed, the odor disappeared in 10 cases, decreased in 3, and remained unchanged in 6; while Hofer claimed total disappearance or marked improvement in all cases. He noticed a marked decrease in crusts in 10 cases, slight decrease in 3, no change in 10, while Hofer claimed almost complete disappearance of the crusts in 90 per cent of the cases. He considers the effect of the vaccine upon the fetor as decidedly the most important, *as the fetor is produced by the Perez organism alone*, whereas the crust formation and the pharyngitis sicca are due in part to the abnormally large nasal cavities. Concerning the atrophic condition, Guggenheim assumes the pessimistic point of view manifestated at Vienna, that no *return to the normal will ever be made possible by the vaccine*. He rather believes in antitoxins than in vaccines and expects the best results from autogenous vaccines, if from any. Dr. Guggenheim's collaborator, Dr. Charles L. Klenk, uses a vaccine composed of the pure culture of the cocco bacillus isolated from the patient's nose. He is certain

that after a time all cases of ozena will be markedly improved and utimately cured. Many of his patients are cured—with the exception of the atrophy. His cures are indeed admirable. Case 1, for instance, shows, although only 3 injections had been given, immediate response to treatment and bids fair to be a cure. Case 8 suffered from unbearable headaches due to her ozena. After 12 injections she felt so fine that she decided to discontinue the treatment. Returned in three weeks with a headache, but no other symptom.

In his preliminary report on his investigations of the vaccine treatment of ozena, Horn is rather conservative. In his second publication, however, he "is extremely sorry for the pessimistic and somewhat suspicious tone which pervaded our former publication and which unfortunately was shared by others who saw the work in Vienna, for our recent work has convinced us that the solution of the etiology of ozena is almost at hand and that very satisfactory results can be hoped for in the treatment of this disease by means of vaccine."

Horn is of the firm conviction that the *cocco bacillus fetidus* of Perez is the only and true cause of ozena. He believes that it has absolutely nothing to do with the bacillus of Abel or other members of the Friedlaender family. Notwithstanding this he considers it convenient to divide clinical ozena into a Friedlaender group and a Perez group, until such time as we can more accurately determine what clinical relation the Friedlaender group has to the cocco bacillus, or what its role is in the etiology of the disease. Most of the investigators and of recent date, especially Burckhardt and Oppikofer, came to the conclusion that the Perez bacillus is closely related to the Friedlaender group.

The identity of the Perez bacillus became more mystified by Perez' statement in 1913, that the cocco bacillus which he had found in ozena secretions exists normally in the nasal cavities of the dog and increases in quantity in dogs that are ill with distemper. He therefore considered the dog as especially capable of spreading the disease.

McGowan, in 1915, proved that there existed a specific organism of distemper which, on account of its resemblance, may be easily mistaken for the organism described by Perez as the cause of ozena. "Distemper (snuffles or sniffles) associated with the presence of the bacillus does not occur only in the dog, but in varying degree of severity also in several other species of animals, including rabbits, which are especially subject to it. In the absence, therefore, of the proof to the contrary, one is justified in provisionally announcing that the rabbits used in Perez' experiments were suffering from

distemper,—it may be in a very mild form—and that cultures made from the nasal discharge subsequent to intravenous injection revealed the presence of the distemper organism which, as one may see from its culture reactions, may easily in a more or less rapic examination be mistaken for the bacillus ozenae fetidus."

Horn and his collaborator, Victors, studied carefully the bacteriology of bacillus bronchisepticus, which was proven by Ferry, McGowan and Torrey and Rahe to be the specific organism of canine distemper. They find the bacillus bronchisepticus to be morphologically almost identical and in many instances biologically similar to the Perez organism. Let us bear in mind (1) that Hofer could not isolate the Perez bacillus from the crusts, but had to inject animals and then isolated it from their nasal discharge; (2) that Hofer adds to his vaccine, in order to make it efficient, a strain from the normal discharge of the dog; (3) that Perez claimed the cocco bacillus to be normally present in the nasal cavities of the dog; (4) that also Horn uses only vaccines whose potency has been increased by passage through animals; (5) that, according to Hofer and Victors, in size, shape, tinctorial reaction and motility the bacillus bronchisepticus and bacillus Perez are almost identical; (6) that grouping according to mitrification is permitted of both organisms; (7) that growth upon potato is similar in most instances. All this evidence points to a possible false conception of the etiology of ozena. Brill's disease was for a long time considered as a clincal entity and was later found to be nothing but typhoid. The slight differences between the almost identical bacillus Perez and bacillus bronchisepticus *may be explained by the adaptation of the animal bacterium to the culture medium of the human nose,* whereby some underlying organic diseases may also be of influence. The bacillus of distemper may therefore be considered as the causative germ of *one type of ozena.* For there is no doubt that there exist both clinically and bacteriologically different types of the disease.

Even Horn and Victors had to acknowledge the frequency with which organisms of the Friedlaender group are isolated from typical and atypical ozena cases. While Burckhardt and Oppikofer and many other investigators placed the Perez bacillus among the Friedlaender group, Horn and Victors are rather inclined to accept the theory of *bacteriological mutation.* They even concede that the cocco bacillus fetidus ozenae group is made up of many subvarieties or strains. The dilemma these investigators are in, becomes evident when we read on the same page the following statements: "Cocco bacillus fetidus ozenae is probably the specific organism of typical fetid ozena in man" (note: probably and in man). On the

other hand: "The cocco bacillus fetidus ozenae Perez, as isolated by Hofer" (note: Hofer had to inject rabbits to obtain the bacillus from the nasal discharge of the animal) "has answered all the bacteriologic requirements necessary to establish its identity as the etiologic factor of ozena." They make, however, the reservation, that they were unable to obtain a complement fixation with untreated oezna cases.

It may be possible that there may be two or more types of ozena, bacteriologically different but clinically identical. In spite of the presence of organisms of the Friedlaender group in cases of undoubted clinical ozena, they consider these organisms as having no etiologic significance. As it is sometimes impossible to isolate the Perez bacillus from the Friedlaender group and, therefore, autogenous vaccine cannot be prepared in these cases, they overcome the difficulty by simply using stock Perez vaccine, whereby almost as brilliant results may be obtained. The authors confess that some cases of ozena showed improvement after the administration of stock-mixed vaccine containing staphylococci, micrococcus catarrhalis, streptococci, etc., but are convinced that these saprophytes clear up as readily with the pure Perez vaccine as they did with the old mixed stock vaccines. Being infatuated with the Perez bacillus theory, Horn and Victors, of course, do not acknowledge any other possible cause of what we call ozena. In their opinion syphilis plays absolutely no role in the etiology of ozena. It would even seem a far cry, in their opinion, to attribute to the accessory sinuses a primary etiologic role. They confess, however, that in order to produce the best results it is absolutely essential, in all cases, to drain the sinuses whenever there is a suspicion of trouble. They are convinced that no amount of vaccine treatment will ever cure a case of Perez infection in the retained pus of an antrum or a sphenoid. There was, therefore, no attempt made to eliminate those cases of ozena from their statistics that had sinus trouble or syphilis, the Wassermann test not being demanded in every case, "because of the expense entailed." The 28 pages of splendid statistics proving the cure or wonderful improvement due to vaccine treatment make very pleasant reading matter, if we bear in mind that the authors differentiate between patients clinically cured and those bacteriologically cured. A bacteriological cure they claim in but one or two cases. And it must be distinctly understood that they have never claimed a case as finally cured. Some of the cases reported are especially interesting. In Case 1 the injection of Shermann's stock vaccine, No. 36, caused on different dates such statements as: crusts better; odor better; crusts decidedly better; odor

much improved; crusts somewhat improved, interwoven by such remarks as: odor marked; odor still strong, etc. Upon the injection of stock Perez vaccine No. 1, we read: odor markedly increased, crusts loosened up; odor entirely disappeared; no crusts; odor still present; crusts diminished; no crusts; no odor; no crusts; slight odor. Treatment resumed after almost a year: Decided odor. No crusts, no odor. Perez bacillus still present.

In Case 5: After 4 injections, Perez stock vaccine No. 6: no crusts, or odor. Two days later: odor strong, nose perfectly clean, except for one crust on the anterior end of the middle turbinate. End of treatment: no crusts, but slight odor. Mucous membrane of nose changed from decided atrophic condition to normal in appearance.

In Case 7: Patient had an extensive double pansinusitis. Innumerable intranasal operations, including double Killian, were done for the relief of discharge and headaches. Had many autogenous vaccine treatments, apparently with little benefit. *One injection mixed Perez vaccine: no more odor, no more crusts.* Two weeks afterwards, with stock Perez injection: nose much improved. The case cleared up wonderfully and has not been seen for several months.

In Case 8: Marked atrophy of both sides; whitish crusts over opening of sphenoids. Marked sweetish odor. Many autogenous injections: decided improvement in crusts; gained three pounds. No odor noticed. Perez bacillus no longer present. *Previous to the treatment with stock Perez vaccines the patient had both frontals, sphenoids, ethmoids and left antrum intranasally opened. Later left sphenoid reopened. Following these operations three injections of No. 6 Perez stock vaccine.* Absolutely no crusts or odor. The authors consider this one of the most brilliant cures of the series. The last sentence of the explanatory note of the case is: Treatment will be omitted until some of the old symptoms develop.

Case 12 is an example of a clinical but not a bacteriological cure.

Case 13 used to complain of headaches, but since he has been washing the nose, does not notice them. Odor and crusts slight at all times. Although he showed a pure Perez and admitted improvement under treatment, he fails to return for his injections.

Case 23, at 18 years of age, noticed odor and smell. Sense of smell and taste lost. Saddle nose, high-grade atrophic condition. Entire nose covered with dark crusts. Odor extremely offensive. No Wassermann test made. This patient, an ignorant Hungarian woman, expressed herself as believing that vaccine treatment was a form of poison and refused further injections, although after the

third injection the odor was decidedly better and the crusts about the same.

Case 24. Abel bacillus. Autogenous vaccine: crusts more moist; crusts the same; no odor; odor and crusts worse; no odor, crusts decidedly improved. Afterwards Perez stock vaccine with marked improvement.

Case 27. Friedlaender and Abel bacillus. Perez stock vaccine: absolutely no odor, few crusts far back. One week later: entire nose filled with crusts, odor strong, but not typical of ozena. Three days later, injection with autogenous vaccine: slight odor, no crusts. One week later: no crust or odor. Eleven days afterwards: odor same, few crusts. The improvement while under treatment was remarkable but often in three or four days the nose would fill up and it would seem that no improvement had taken place. The patient was very stubborn and hard to handle and finally refused further treatment.

In order to control the claims made by Hofer, Guggenheim and Horn, I undertook at the Ear, Nose and Throat Department of Vanderbilt Clinic an investigation in which I was encouraged and provided with material by Prof. Coakley and aided by Dr. Pratt. Miss Pauli and Miss Baker, from Prof. Zinsser's Bacteriological Department of Columbia University, made the bacteriological examinations and prepared the vaccines. I am greatly indebted to Prof. Coakley and Prof. Zinsser and my above collaborators for the valuable encouragement and aid given me in these investigations.

The aim of the investigation was a purely clinical one: to prove or disprove the therapeutic value of the ozena vaccines in vogue. The following types of vaccine were used: Hofer's original, made from Perez bacillus strains and brought by myself from Vienna; autogenous vaccine; polyvalent vaccine prepared from seven strains of our patients; stock vaccine Perez (Horn); mixed Perez vaccine (Horn); Parke Davis ozena vaccine Perez; Parke Davis ozena vaccine combined.

On each patient applying for treatment a Wassermann test was made. If it was positive, the patient was excluded from the vaccine treatment. Wherever there was a suspicion of tuberculosis or sinus involvement, the patient was also refused treatment. The intention was to inject if possible only so-called idiopathic cases of ozena.

The bacteriologic examination and the preparation of the vaccine was done in strict accordance with the methods of Hofer and Horn.

In our method of vaccine administration we followed as closely as possible Horn's directions. In some cases, especially those

treated by the Viennese vaccine and by autogenous vaccine, Hofer's method was adhered to. The crust of each ozena was treated bacteriologically according to the rules laid down by Hofer and the vaccines prepared according to him. Rabbits were injected with the different cultures in order to control the claims made by Hofer and his followers. My statistics lack enthusiastic exaggeration. They report facts as seen by an unbiased observer.

The stock vaccine sent to me by Horn was used in 5 cases; three times with negative result, in one case showing slight improvement in odor, and in another one, in addition to this, a loosening of the crusts. These results were, however, only transient, for after a period of non-treatment the symptoms recurred unabated.

Hofer's original vaccine was used in 2 cases with negative results.

Autogenous vaccine was applied 12 times. In 1 case there was transient improvement, while the remaining 11 showed an absolutely negative result.

Parke Davis vaccine combined was used 5 times, with negative result in 3 cases, and slight transient improvement in 2 cases. Their ozena vaccine Perez was used in one case with negative result. One case had been previously treated by a mixed vaccine of staphylo- and streptococci with negative result.

The bacteriological examination gives the following results: Out of 17 cases injected there was demonstrable in the nasal crusts: Bacillus Perez, 5 times; bacillus Abel, once; bacillus Friedlaender 3 times; pure staphylococci, twice; staphylococci and Gram negative bacillus, twice; staphylococci and Friedlaender bacillus, twice; micrococcus catarrhalis, once.

The pathological changes on the turbinated of the injected rabbits showed nothing characteristic of ozena. The only changes noticeable were congestion and slight discharge. Clinically, the injected rabbits resembled very much those suffering from distemper.

In conclusion I wish to say that in my opinion ozena, as characterized by foul odor and yellowish crusts, is no clinical entity and is therefore not caused by a specific organism such as the Perez bacillus. In those cases where the cocco bacillus fetidus Perez is found, there has been transmitted directly or indirectly from the animal's nose (dog, rabbit, etc.) the bacillus bronchisepticus, the causative germ of distemper, upon a previously predisposed soil of atrophied and, therefore, abnormally functionating nasal mucous membrane. The Perez bacillus has never been found in a normal nose and even not in one showing atrophy without crust formation and fetor. Crustation is due to the stagnation of secretion in an enlarged cavity. Only within these decomposing crusts, presenting an offensive odor,

have those bacilli been found (and of course from there they might spread all over the nose and the adjoining accessory cavities) that have been claimed by different investigators as being the etiological factors of ozena. Here we find the Perez bacillus (the transplanted bacillus bronchisepticus), the Abel and Friedlaender bacilli, the pneumococcus, micrococcus catarrhalis, bacillus pyocyaneus, staphylococci, etc., etc. It seems that the decaying crusts form a suitable soil for these germs to grow upon. *Through their growth these different bacilli increase, of course, the putrefactive process and through it the stench and crustation. It is therefore plausible that if vaccine treatment is of any scientific value at all it may relieve, when applied in ozena, that small part played in the increase of the symptoms of crustation and fetor by the action of the particular germ from which the vaccine is taken. We, therefore, find transient improvement in crust formation and fetor not only by the much heralded Perez vaccine, but also by any stock vaccine of staphylococci, streptococci, Abel bacillus, Friedlaender bacillus, etc., etc., if these germs happen to be present in the crusts.* Strong antiseptic and germicidal local applications after thorough and regular cleansing will have a similar transient positive effect upon both crusts and fetor. The crux in the treatment of what we call ozena lies in the fact that the atrophy of the nasal mucous membrane and its underlying structures is the primary condition and that this atrophy will therefore never be cured by any vaccine treatment whatsoever, as agreed upon even by the most ardent vaccine advocates. The primary atrophic condition is causd by bad atmospheric and hygienic circumstances, by chronic general ailments such as tuberculosis, syphilis, anemia, metabolic disturbances, toxemias, etc. Prophylaxis should be the watchword, as long as a cure is unavailable. Let us try to avoid and cure all conditions which lead to the atrophy of the nasal mucous membrane. Once the atrophy becomes a chronic one and shows the complication of decaying crusts, therapy will be symptomatic only and will give no chance of a cure, whether the treatment consists of paraffine injection, galvanization, application of antiseptics, scarlet red, etc., or of vaccine injection. It is a proven fact that the injection of any kind of fluid improves, for a time, subjectively the patient's condition. Objectively, we are human beings all of us, including the vaccine adherers, and sometimes, therefore, we may see the crusts and smell the fetor with our centers of vision and of smell, unconsciously befogged by the intense desire for positive vaccine achievements. With due credit to the enthusiastic endeavors of our European and American confrères, there is no doubt in my mind that ozena will outlive its vaccine treatment.

CASE REPORTS.

Case 1. L. B. L., aged 21 years, female. *Family history* negative. *Past history:* For past three years foul odor from nose, combined with greenish crusts. Loss of smell. Never played with dogs.

Examination. Excessive atrophic condition of both nasal cavities, greenish foul smelling crusts covering inferior and middle turbinates and partly the septum. *Wassermann test* negative.

Bacteriological examination. March 2, 1916, at 3 p. m., crust removed from right middle turbinate and dropped into sterile test tube; 9 p. m., entire crust dropped into 2 per cent glucose broth.

March 3, 1916, 9 a. m. One loopful of broth culture spread over 2 per cent glucose agar plates, also streak plates made on ascitic agar and plates incubated at 37° C. for 24 hours.

March 4, 1916. No odor on plates. Colonies fished to glucose slants.

March 10, 1916. Culture dead.

Treatment. April 15, 1916, first injection (Perez stock, 50 million; Horn). One hour later slight headache and dizziness. April 16, 1916, 100 million. Patient states that her people "already noticed an improvement in odor." Objectively no change. April 17, 1916, 200 million. Slight general reaction; headache, dizziness, sleeplessness, lack of appetite. Local reaction very marked. April 20, 1916, 400 million. Much discharge from nose, but otherwise no change. April 23, 1916, 600 million. Much dizziness, stiff arm, fever. April 26, 1916. Patient reports that for two days her people noticed no odor at all but to-day the odor returned. No injection. April 29, 1916, 800 million. Objectively crusts and odor decreased. May 7, 1916, 1 billion. Good appetite, feels improved. May 14, 1916, 1 billion and 200 million. Less crusts, but odor very bad. May 21, 1916, 1 billion and 400 million. Patient claims there is a decrease in crusts and odor, noticeable also objectively. May 28, 1916, 1 billion and 600 million. Continued improvement. June 5, 1916, 1 billion and 800 million. Continued improvement. June 13, 1916, 2 billion. Slight amount of crusts. Recurrence of odor. June 20, 1916, 2 billion and 200 million. Same condition. June 27, 1916, 2 billion and 400 million. Few crusts, odor present but not offensive.

After this last injection the patient reported every two weeks. One week afterwards the crusts and the offensive smell recurred and became gradually worse, until at the end of July the patient complained that the amount of crusts and the offensiveness of the odor were worse than ever before. The patient went to the country

and in her letters written at regular intervals of one week she complained that the crusts and the odor were unbearable.

October 20, 1916. The entire nasal cavity covered by tenacious greenish crusts, foul odor detectable at three and one-half feet. A crust was taken in the regular way and examined bacteriologically. Bacillus Perez was found. Autogenous vaccine was prepared.

Nov. 3, 1916. Injection of autogenous vaccine, 100 million. Nov. 6, 1916, 200 million. No general reaction, slight local reaction. Nov. 9, 1916, 300 million. Patient thinks that the amount of crusts has decreased. Nov. 12, 1916, 400 million. General reaction: dizziness and fever. Nov. 19, 1916, 500 million. Objectively no change, but patient claims improvement in the amount of crusts. Nov. 24, 1916, 600 million. Amount of crusts slightly decreased. Nov. 26, 1916, 700 million. Odor less noticeable. Nov. 29, 1916, 800 million; Dec. 3, 1916, 900 million; Jan. 2, 1917, 1 billion. Crusts and odor improved.

The patient returned Jan. 5, 1917, and complained that both the odor and the crusts were just as bad as before any vaccine treatment was started. Objectively, large greenish crusts of sharp odor were seen covering the inferior and middle turbinates, the odor was detectable at three feet. The patient declined any further vaccine treatment. Irrigations with weak tincture of iodin solution (15-20 drops to one-half glass of warm water) advised. Three weeks later the patient claimed to feel markedly improved. On March 15, 1917, under continuous irrigations with tincture of iodin solution the turbinates were covered by thin mucus that gave only a very faint odor. No crusts.

Case 2. F. Y., aged 20 years, female.

Family history. Sister suffers from the same trouble.

Past history. Bad smell and crusts from the nose since 7 years of age. Had several nasal operations performed to improve the condition and was treated locally as well as by injections of staphylococcus and streptococcus mixed vaccine, without avail.

Examination. Dark greenish crusts of intense odor covering the turbinates and septum. Very marked atrophy. Dry pharyngitis and laryngitis.

Jan. 22, 1916. Treatment with vaccine made from Hofer's original Perez bacillus strain which I brought from Vienna. Dose, 50 million.

Jan. 29, 1916, 100 million; Feb. 5, 1916, 150 million; Feb. 12, 1916, 200 million, no reaction yet; Feb. 18, 1916, 250 million; Feb. 25, 1916, 300 million, slight fever, marked local reaction.

March 3, 1916, 350 million. No change in crusts or odor.

March 9, 1916, 400 million; March 16, 1916, 450 million; March 23, 1916, 500 million; March 30, 1916, 600 million, crusts and odor unchanged.

April 23, 1916, treatment with ozena vaccine combined (Parke, Davis Co.) was started, and on April 24, 1916, 50 million injected. Patient complained of headache, weakness, dizziness, lack of appetite, fever, restlessness, attacks of vomiting. Local reaction very severe: purple discoloration and pain. Patient claims that the crusts in the nose and less, was sneezing all day long. Had washed the nose twice during the day; in the evening thick crusts came out, without odor.

April 27, 1916, 100 million. Less crusts, sense of smell improved. No headache.

April 29, 1916, 200 million. Very many crusts; slight odor.

May 1, 1916, 400 million; May 3, 1916, 600 million; May 5, 1916, 800 million; May 7, 1916, 1 billion. Patient feels much improved. Almost no odor and hardly any crusts.

May 9, 1200 million; May 11, 1400 million; May 13, 1600 million; May 15, 1800 million, almost no crusts, slight odor; May 17, 2 billion; May 19, 2200 million; May 21, 2400 million; May 23, 2600 million; May 26, 2800 million; June 1, 3 billion; June 4, 3200 million; June 8, 3400 million. Odor slightly noticeable; few crusts. Patient states that she feels markedly improved. She returned on Oct. 1, 1916. During the summer months the condition of the nose had gradually become aggravated until at the present time the crusts and the odor were just as bad as before treatment.

Oct. 8, 1916, treatment with autogenous vaccine was started, and 100 million injected. Oct. 11, 200 million; Oct. 14, 300 million; Oct. 17, 400 million; Oct. 18, 500 million. Crusts and odor unchanged. Oct. 22, 600 million; Oct. 24, 700 million; Oct. 28, 800 million. Many greenish crusts of offensive odor. Patient discontinued treatment.

Bacteriological examination. March 23, 1916. Entire crust was removed from the right inferior turbinate and dropped into 2 per cent glucose broth. The next day a series of fine glucose agar plates were streaked with a rod dipped in the broth culture and inoculated for 24 hours. March 22, very marked, sickening odor from the plates. Colonies fished to glucose agar slants, which after nine hours gave off a characteristic strong ozena odor.

March 23, odor from slants very slight.

March 24, odor entirely disappeared.

Rabbit No. 471, weight 1325 grams, inoculated intravenously with 1 c. c. of living 24-hour culture (1 loop culture in 5 c. c. salt solution).

March 25, rabbit in good condition. March 26, rabbit seems to have slight difficulty in breathing.

March 29, rabbit weighs 1103 grams (loss of 222 gms.). Inoculated with one-half c. c. whole slant emulsified in 2 c. c. salt solution.

March 30, loss of appetite. April 1, rabbit seems very sick; nostrils congested, difficulty in breathing. Weight, 995 gms. (loss of 330 gms. since first injection).

April 3, slight discharge from nose. April 5, rabbit apparently recovering. April 11, weight of rabbit 815 gms. (loss of 510 gms.). Inoculated with 2 c. c. of the culture, April 29. During this period the rabbit seemed apparently well and lively, only that he would not eat. Rabbit died. Cultures negative.

Case 3. V. R., 18 years old; female.

Family history. Mother suffers from similar nose trouble. *Past history:* Since three years past crust formation and bad odor growing worse during the last six months. Headaches, dry feeling in the throat. Fondness for dogs.

Examination. Entire Schneiderian membrane in atrophic condition. Wassermann test negative. Inferior and middle turbinates, also part of the septum, covered with dark crust of offensive odor.

Bacteriologic examination same as in Case 2. Perez bacillus.

April 10, 1916, injection with Parke, Davis Co. ozena vaccine (Perez), 50 million. April 12, 100 million; April 15, 200 million. Marked local reaction. Dizziness, dull headache, weakness. April 18, 400 million; April 21, 600 million. Severe headache, swelling of injected arm; no crusts or odor.

April 24, 800 million; April 27, 1 billion; April 30, 1200 million. No change.

May 2, 1400 million. Patient feels worse. More crusts; odor worse.

May 5, 1600 million; May 12, 1800 million; May 19, 2 billion. Patient still complains that the condition of her nose is getting worse.

May 26, 2200 million; June 6, 2400 million; June 9, 2600 million. The patient felt very much dissatisfied and discontinued treatment. Local examination at this time showed the entire nasal cavity on both sides to be covered by discolored, thick, adherent crusts of intense, characteristic odor.

The patient returned to the Clinic on November 3, 1916. Bacteriologic examination again revealed B. Perez. An autogenous

vaccine was made. The condition of the nose was as bad as before.

Nov. 18, 1916, injection of 100 million autogenous vaccine; Nov. 25, 200 million; Dec. 2, 400 million; Dec. 7, 600 million. Odor and crusts unchanged. Dec. 12, 800 million; Dec. 19, 1 billion. Crusts and odor unchanged. Patient discontinued vaccine treatment. She was told to use irrigations with a weak solution of tincture of iodin. She returned Feb. 15, 1917. The crusts were fewer and more easily removable, but the odor had not changed.

Case 4. Mrs. V. M., mother of the former patient, 44 years old. *History:* Continuous headache for past year, loss of sense of smell, dry throat, difficulty in swallowing, purulent discharge. No crusts, no bad odor. Wassermann negative.

Bacteriologic examination same as in Case 3. No characteristic germ found.

Examination shows pus under the middle turbinates which, together with the frontal tenderness and the findings with transillumination, points to involvement of the frontal sinuses. There is a marked atrophic condition of the inferior turbinates. No crust formation and no odor.

As this case of atrophic rhinitis was not a genuine one, no vaccine treatment was undertaken.

Case 5. Mrs. N. V., 35 years old. *Family history* negative. *Past history:* severe headaches, muco-purulent discharge, crust formation, for many years. Severe odor at times. Wassermann negative.

Bacteriologic examination the same as in the other cases, only with negative result. Staphylococci in nearly pure culture.

Examination. Deviation of septum to the right; hypertrophy of the right inferior turbinate; left nasal cavity atrophic; inferior and middle turbinates covered by small tenacious crusts with slight but not offensive odor; pressure pain over the left frontal sinus and ethmoidal cells. Transillumination doubtful.

This case of atrophic rhinitis could be traced to sinus involvement and was therefore not subjected to vaccine treatment.

Case 6. R. G., female, 14 years old. *Family history* negative, except that her mother complains of nose trouble.

Past history. Patient suffers for many years from bad nasal odor and crust formation. Was never fond of dogs. Had to wash her nose twice daily. One year ago she underwent a nasal operation. No sense of smell.

Examination. Deviation of septum to the left. Atrophy of the entire nasal cavity. Right inferior and middle turbinates, part of

septum and naso-pharynx covered by thick, yellowish, tenacious crusts, with slight odor only. Wassermann negative.

Bacteriologic examination. April 7, 1916, a whole crust was removed from the right inferior turbinate and placed in 2 per cent glucose broth for 14 hours. A rod was dipped in the culture and smeared over the surface of five glucose agar plates which were incubated at 37° C. for 48 hours. Colonies were fished and transferred to glucose agar slants. No odor was present, although a very putrefactive odor arose from the colonies on the plates, but which was not characteristic. Perez bacillus present.

Animal experimentation. April 11, 1916, an entire 24-hour agar slant was emulsified in 1½ c. c. saline and rabbit No. 777 was inoculated with 1¼ c. c. of the entire slant. Weight of the rabbit was 970 grams.

April 12, 1916, the rabbit was found dead in the cage. Necropsy showed normal conditions in the nose.

May 9, 1916, rabbit No. 42 was inoculated with 2 c. c. of living culture. Weight of rabbit, 1435 gms.

May 10, 1916, rabbit dead. Necropsy negative.

Treatment with autogenous vaccine. April 22, 1916, 50 million; April 23, marked local reaction, 100 million; April 24, 200 million; dizziness and headaches; April 25, 300 million; April 27, 400 million; April 30, 600 million; May 3, 800 million; May 7, 1 billion. During the entire treatment there was no change in the crust formation. Odor slightly decreased.

May 12, 1200 million; May 17, 1400 million. No change. Patient feels very bad and complains of headaches and dizziness. Treatment interrupted.

In October, 1916, the patient returned for re-examination. Subjectively and objectively, the same local condition as before the vaccine treatment.

Case 7. L. U., female, 21 years old. *Family history:* One sister suffers from a similar nasal condition. *Past history:* Headaches, weak eyes, nervousness, anemia, strong odor from the nose, crust formation, dry throat, frequent bronchitis for 10 years. Was several times previously inoculated with different vaccine, without avail however.

Examination. Both nasal cavities in a state of marked atrophy. Turbinates, septum and nasopharynx covered by thick, dark green crusts with offensive odor, noticeable at five feet. Wassermann four plus positive.

This case of atrophic rhinitis was not subjected to vaccine treatment as there was a suspicion of luetic origin.

Case 8. G. U., sister of Case 7. *Past history:* Severe headaches, strong odor and crust formation for two years.

Examination. Extreme atrophy in both nasal cavities; slight crust formation, the entire mucous membrane covered by muco-purulent discharge; slight odor. Moist Laryngitis; hypertrophic tonsils. Wassermann, though not strongly positive, mode of agglutination suspicious of lues.

Bacteriologic examination. Crust from nose treated as in the other cases.

May 9, 1916, rabbitt No. 93, weighing 1543 gms., inoculated with 2 c. c. of broth culture. May 16, inoculated with one-quarter slant. May 19, rabbit dead. Autopsy and cultures negative.

This case of atrophic rhinitis was not treated by vaccines for the same reason as in the preceding case. These two cases were referred to the Clinic by Dr. I. M. Heller.

Case 9. T. K., male, 27 years old. *Family history* negative. *Past history:* For over 10 years foul odor and crust formation in both nostrils. Never fond of dogs.

Examination. Slight deviation of the septum. Both inferior and middle turbinates markedly atrophic, covered by thick, greenish crusts of characteristic odor. Crust formation in the nasopharynx. Wassermann negative.

Bacteriologic examination. Whole crust was removed and placed in 2 per cent glucose agar broth for 14 hours, then a rodful was smeared over the surface of five plates, sugar, acid and gas on maltose and dextrose. Bacillus Abel.

May 9, 1916, rabbit No. 61, weighing 1550 gms., inoculated with 2 c. c. living culture. May 16, inoculated with one-quarter slant. May 20, rabbit well. No nasal changes.

Vaccine treatment. April 15, 1916, initial dose of 50 million Horn's mixed vaccine (seven different strains). April 16, 100 million, marked local reaction; April 17, 200 million; April 18, 400 million, slight headache; April 19, 800 million. Patient's people claim that the odor is less offensive. April 20, 1 billion, severe headache, no change objectively in crusts or odor. April 23, 1200 million; April 26, 1400 million; April 29, 1600 million; April 30, 1800 million; May 3, 2 billion, absolutely no change and patient feels generally weak. May 7, 2200 million; May 11, 2400 million; May 15, 2600 million; May 19, 2800 million; May 24, 3 billion; May 27, 3200 million.

At this time the amount of crusts and their character had not changed and the offensiveness of the odor was also the same. The patient asked for a change in the treatment and the injection of

autogenous vaccine was suggested. The patient returned Dec. 15, 1916. The condition of his nose was unchanged. He decided to postpone the treatment with autogenous vaccine for the present.

Case 10. H. C., female, 20 years old. *Family history* negative. *Past history:* Occasional bad odor from the nose, no crusts but much muco-pus and headaches for one and one-half years.

Examination. Both middle turbinates hypertrophied, yellowish pus from beneath them. Transillumination shows slight cloudiness of both frontal sinuses. Beginning atrophy of both inferior turbinates. Wassermann negative.

Bacteriologic examination negative as to Perez or Abel bacilli. Micrococcus catarrhalis present.

This case of beginning atrophic rhinitis, owing to its apparent origin from the frontal sinuses, was not subjected to vaccine treatment.

Case 11. M. L., male, 79 years old. *Family history* negative. *Past history:* For two years crust formation and bad odor; dryness in the throat.

Examination. Deviation of the septum to the right. Marked atrophy of entire left nasal cavity. Left inferior and middle turbinates covered by thick, greenish crusts with offensive odor. Dry pharyngitis. Wassermann negative.

Bacteriologic examination. The previously described method revealed streptococci and a Gram negative bacillus, also staphylococci. Sugar reactions: acid and gas on dextrose, mannite, saccharose; unchanged on lactose.

Animal experimentation. May 9, 1916, rabbit No. 76, weighing 1240 gms., inoculated with 2 c. c. broth culture. May 10, rabbit dead. Necropsy and cultures negative.

Vaccine treatment. May 13, 1916, injection 100 million Parke, Davis Co. ozena vaccine combined. May 16, 200 million; marked local reaction. May 19, 400 million; May 22, 800 million. No changes noticeable. May 25, 1800 million. Slight general reaction the following day: uneasiness and headache. May 28, 2 billion. Crusts somewhat thinner; odor very offensive; general weakness. June 10, 2800 million; June 14, 3 billion; June 18, 3200 million; June 22, 3400 million. Patient feels very weak; crusts numerous, odor offensive.

Treatment was discontinued and the patient was told to irrigate his nose with weak solution of tincture of iodin. Four weeks later he reported no change in the amount and quality of the crusts; slight decrease in odor. Objectively the same condition as before treatment.

Case 12. G. M., female, 46 years old. *Family history* negative. *Past history:* Crusts with bad odor from right side of the nose for one year.

Examination. Deviation of the septum to the left side. Right nasal cavity markedly atrophic, especially the middle turbinate. Greenish crusts on middle turbinate, moderate in amount, but with characteristic odor. Wassermann negative.

Bacteriologic examination. Great abundance of staphylococci and a Gram negative bacillus.

Animal experimentation. May 9, 1916, rabbit No. 45, weighing 1555 gms., inoculated with 2 c. c. broth culture. May 9, inoculated with one-quarter slant. May 12, rabbit well; no nasal discharge.

Treatment with autogenous vaccine. May 12, 100 million; May 14, 200 million, marked local reaction; May 17, 400 million, general reaction; May 21, 600 million, odor decreased; May 25, 800 million; May 29, 1 billion, odor again as bad as before, crusts somewhat thinner; June 3, 1200 million; June 7, 1400 million; June 11, 1600 million; June 15, 1800 million; June 20, 2 billion, crusts again thick, odor unchanged; June 25, 2200 million; June 29, 24,000 million.

On account of negative result treatment was discontinued.

Case 13. Ç. A., female, 11 years old. *Family history* negative. *Past history:* For two years yellowish crusts few times daily from both nostrils. No odor.

Examination. Total atrophy of both inferior and middle turbinates, also of the mucous membrane of the septum. Entire mucous membrane of the nose and naso-pharynx covered by thick, greenish-yellow crusts with but slight odor. Pharyngitis sicca. Wassermann negative.

Bacteriologic examination. Same procedure as in the preceding cases. Almost pure culture of staphylococci and a small Gram negative bacillus on plates. Gas and acid formation on dextrose, maltose and lactose; no change on saccharose.

Animal experimentation. May 9, 1916, rabbit No. 97, weighing 1475 gms. inoculated with 2 c. c. broth culture. May 16, inoculated with one-quarter slant. Rabbit remains well. No nasal discharge.

Treatment with autogenous vaccines. May 16, 1916, 50 million; May 18, 100 million; May 20, 200 million, marked general reaction. May 23, 400 million; May 27, 600 million; May 31, 800 million. No change in crusts, patient feels generally weak. June 4, 1 billion; June 8, 1200 million. Crusts slightly softer but not reduced in quantity. June 14, 1400 million; June 20, 1600 million; June 27, 1800 million; July 2, 2 billion. Crusts again thicker; odor slightly

increased. July 9, 2200 million; July 16, 2400 million. Treatment discontinued.

The patient returned Sept. 20, 1916. The condition of the nose had not changed, except that the odor became more pronounced. Further vaccine treatment was refused by the parents. Irrigations with weak tincture of iodin solution was advised. Dec. 10, 1916, the patient returned, with the statement that the crusts are removable more easily and that between the irrigations she feels quite "clan and comfortable." The crusts appear looser.

Case 14. D. M., female, 20 years old. *Family history* negative. *Past history:* For six years marked crust formation with offensive odor, which requires washing the nose about five times a day. Loss of smell. Dry throat.

Examination. Marked deviation of the septum to the left. Total atrophy of the right nasal cavity. Thick greenish crusts of offensive odor covering the right middle and inferior turbinates. Wassermann negative.

Bacteriological examination revealed bacillus Perez.

Treatment by injections of mixed Perez vaccine (Horn). May 11, 1916, 100 million; May 14, 200 million, marked local reaction. May 17, 400 million; May 20, 800 million; May 23, 1600 million; May 28, 3200 million. General reaction with high fever, severe headache, dizziness, malaise. June 2, 3400 million; June 6, 3600 million. Relatives claim to notice less odor. Objectively, some decrease in odor. June 10, 4 billion; June 14, 4400 million. Crusts somewhat looser but not reduced in quantity. Patient washes the nose three times a day. June 20, 4800 million; June 27, 5 billion; July 4, 5200 million. No further change was noticeable since June 14. Treatment interrupted.

The patient returned two months afterwards with the statements that the crusts have returned to the condition before treatment and that she must again wash her nose five times daily. The only difference noticeable is a less offensive odor. January, 1917, the patient again noticed an aggravation of the odor, but it was still slightly better than before treatment. The crust formation had remained unchanged.

Case 15. L. M., female, 55 years old. *Family history* negative. *Past history:* Crust formation, bad odor, frequent nasal bleeding, headaches, loss of smell of six months' duration.

Examination. Both nasal cavities totally atrophic and covered by very thick, dry crusts of offensive odor, noticeable at five feet. Wassermann negative.

Bacteriologic examination negative as to Perez and Abel bacilli. Bacillus Friedlaender present.

Treatment by injections of Parke, Davis ozena vaccine combined. May 11, 1916, 100 million; May 14, 200 million, local reaction. May 17, 400 million; May 21, 800 million; May 25, 1600 million. General reaction. May 29, 1800 million. No change in crusts or odor. June 2, 2 billion; June 6, 2200 million. Odor noticeable only at 4 feet. Crusts unchanged. June 10, 2400 million; June 14, 2600 million. Crusts changed from dark into dark greenish in color. Odor still to be smelled at four feet. June 18, 2800 million; June 24, 3 billion. Odor again noticeable at five feet. June 28, 3200 million. Patient still has nose bleeds two or three times a day. The color of the crusts has turned into greenish-yellow. July 2, 3400 million. No further change in crusts or odor. Treatment interrupted.

The patient returned Oct. 2, 1916. Odor very offensive, noticeable at 5½feet; crusts dark greenish; removal followed by bleeding.

Case 16. B. A., female, 17 years old. *Family history:* Mother and sister suffer from similar trouble. *Past history:* Headaches, crust formation and bad odor in nose for eight years. Operated several times for relief of the trouble without success.

Examination. Both inferior turbinates atrophic and covered by thin, dark greenish crusts with offensive odor. Both middle turbinates hyptertrophic with pus oozing from beneath them. Marked pressure pain in region of frontal and ethmoidal sinuses. Transillumination negative. Wassermann positive.

Bacteriologic examination. Staphylococci.

Although in every case where there was a suspicion of syphilis or sinus involvement vaccine treatment was not given, an exception was made in this case as the patient, having seen others receiving injections, asked "to be also given a chance."

Treatment. June 18, 1916, injection of Parke, Davis & Co. ozena vaccine combined, 100 million; June 21, 200 million; June 23, 600 million; June 26, 800 million; June 30, 1200 million. No change. July 4, 2400 million; July 7, 3 billion, slight general reaction. July 11, 3200 million; July 14, 3400 million; July 18, 3600 million; July 21, 3800 million.

As no change occurred as to crusts or odor, treatment was discontinued.

Case 17. M. R., female, 20 years old. *Family history* negative. *Past history:* Crusts and bad smell for three years, slight cough, hoarseness and recently dysphagia.

Examination. Total atrophy of both nasal cavities; abundance of greenish, thick crusts of offensive odor. The nose represents the typical clinical picture of ozena. Laryngeal examination reveals a turban-shaped infiltration of the epiglottis, with ulcerations on its inner surface. As this suggested tuberculosis as the underlying cause the patient was referred to the tuberculosis department where the following report was given: Broncho-vesicular respiration at the right apex; anteriorly and posteriorly, sibilant rales over the right upper lobe. Diagnosis: Pulmonary tuberculosis.

Tuberculosis being the underlying cause this case of atrophic rhinitis was not treated by vaccine.

Case 18. F. G., female, 7 years old. *Family history* negative. *Past history:* Crust formation and bad odor in the nose, of nine months' duration; also dryness of the throat.

Examination. Very pronounced atrophy of both nasal cavities. Over the right inferior and middle turbinates and over the left inferior and middle turbinates and over the left inferior turbinate, yellowish crusts with offensive odor that can be smelled at six feet. Wasserman negative.

Bacteriologic examination: Staphylococci and bacillus of Friedlaender.

Treatment. April 15, 1916, injection with mixed Perez vaccine (Horn), 50 million; April 16, 100 million; April 17, 200 million, very marked local reaction. April 18, 400 million; April 19, 800 million; April 20, 1 billion. Marked general reaction: weakness, fever and headache. (From Jan. 22, 1916, to March 26, 1916, the patient was treated with increasing doses of autogenous vaccines up to 2 million without any effect on the condition of the nose).

April 23, 1200 million, crusts decreased, odor less offensive. April 27, 1400 million; April 30, 1600 million. General condition weakened. Inspection of the nasal cavities shows the presence of thickened pus in place of crusts, odor noticeable at only four feet.

May 3, 1800 million; May 7, 2 billion; May 11, 2200 million; May 15, 2400 million. Odor noticeable at three feet only. Tendency to crust formation again, but the crusts are more easily removable. May 19, 2600 million; May 23, 2800 million; May 27, 3 billion. No further change.

June 1, 1916, 3200 million; June 4, 3400 million; June 10, 3600 million. Relatives believe the odor to be much less noticeable. Objectively, however, it can still be detected at a distance of three feet. It is less offensive. The crust formation is restricted to the inferior turbinates; there are thick aggregations of muco-pus in the nasopharynx. Treatment interrupted.

The patient returned Oct. 12, 1916. She is bothered by the crust formation just as before the treatment. The bad odor is, however, less noticeable. Objectively, the odor can be detected at three feet. There are yellowish crusts covering both inferior turbinates rather loosely. When re-examined, Jan. 18, 1917, the same condition was present.

Case 19. C. W., female, 26 years old. *Family history:* Mother suffers from the same trouble. *Past history:* Crust formation and bad odor since childhood. Was operated upon several times in the nose without relief.

Examination. Both middle turbinates missing; signs of operative interference on both ethmoidal cavities. Mucous membrane of both nasal cavities highly atrophic. Both inferior turbinates, part of the septum and nasopharynx covered by tenacious crust of dark, green color and offensive odor that can be detected at four feet. Wassermann negative.

Bacteriologic examination. Friedlaender bacillus and staphylococci.

Treatment. Injection with Perez vaccine (original of Hofer brought by myself from Vienna). Feb. 12, 1916, 50 million; Feb. 19, 100 million; Feb. 26, 200 million; March 3, 400 million; March 10, 800 million; March 17, 1600 million. Absolutely no reaction or change. March 24, 2 billion; March 30, 2400 million; April 6, 2400 million; March 13, 3 billion. No change. Treatment interrupted.

April 17, 1916, injection with Parke, Davis & Co. ozena vaccine Perez, 50 million; April 18, 100 million; April 19, 200 million; April 20, 400 million; April 21, 800 million. Marked local reaction, also headache, dizziness, weakness. April 24, 1 billion. Crusts somewhat looser, no change in odor. April 27, 1200 million; May 1, 1400 million; May 4, 1600 million; May 4, 1800 million. No changes. May 8, 2 billion. Patient complains that the crusts are getting thicker and cannot be removed easily; purulent discharge. May 14, 2200 million; May 18, 2400 million. At times, the patient thinks that she feels slightly better, but at other times she thinks her condition to be about the same. Objectively, no change is noticeable.

May 23, 2600 million; May 27, 2800 million; June 1, 3 billion; June 5, 3200 million; June 9, 3400 million; June 15, 3600 million. During the last two weeks the patient states that she has felt worse and must wash her nose more frequently. Treatment interrupted.

The patient returned on November 3, 1916. During the last two months the crust formation had increased. The odor had remained about the same. Bacteriologic examination showed the bacillus of Friedlaender in pure culture.

Injections with autogenous vaccine started Nov. 19, 1916, and continued to Jan. 22, 1917, up to a dose of 3400 million germs, showed no change whatever.

Case 20. W. R., male, 16 years old. *Family history* negative. *Past history:* Crust formation in the nose with bad odor for six years; patient has to wash his nose three times a day.

Examination. Total atrophy of mucous membrane of the right nasal cavity, beginning atrophy of the left nasal cavity. Right nasal cavity filled with yellowish, thick crusts, similar ones on left inferior turbinate; pharyngitis sicca. Wassermann negative.

Bacteriologic examination. Perez bacillus.

Treatment by injections of autogenous vaccines. Feb. 18, 1916, 100 million; Feb. 24, 200 million; Feb. 28, 400 million; March 3, 800 million; March 7, 1600 million, general reaction. March 11, 1800 million; March 14, 2 billion, absolutely no change. March 18, 2200 million; March 24, 2400 million; March 29, 2600 million; April 3, 2800 million. No change whatever. Treatment interrupted.

April 15, 1916, treatment started with mixed stock vaccine Perez (Horn), 50 million; April 16, 100 million; April 17, 200 million, positive general reaction: headache, loss of appetite, dizziness, weakness. April 21, 400 million; April 23, 600 million, general weakness continues, no local change; April 27, 800 million; May 1, 1 billion; May 4, 1200 million; May 7, 1400 million. Slightly less crusts, noticeable objectively also. Odor unchanged. May 11, 1600 million; May 15, 1800 million; May 19, 2 billion. Crusts again bad. May 23, 2200 million; May 27, 2400 million. Patient has been told by friends that the odor is less noticeable, but it can still be detected at three feet.

June 1, 2600 million; June 5, 2800 million. Condition of crusts unchanged. Odor at three feet. June 9, 3000 million; June 15, 3200 million; June 19, 3400 million; June 24, 3600 million.

No further changes. Treatment discontinued. Patient returned Nov. 6, 1916. The odor had recently become even more offensive and was to be detected at six feet. The left inferior turbinate has also become atrophic and covered by yellowish crusts with bad odor.

Case 21. B. L., female, 22 years old. *Family history* negative. *Past history:* Foul odor from the nose, with many crusts for five years. Numerous nasal operations performed without relief.

Examination. In both nasal cavities, inferior and middle turbinates previously removed. Atrophy of the highest degree; thick, yellowish crusts covering entire mucous membrane; very offensive odor. Wassermann negative.

This case of atrophic rhinitis having been caused by the operative removal of physiological organs, was not subjected to vaccine treatment.

Case 22. B. G., female, 17 years old. *Family history* negative. *Past history:* When 10 years of age a radical mastoid operation was performed on the right side. One year ago a post-auricular sinus opened, with continuous discharge. At the same time crust formation occurred in the nose and a bad odor developed.

Examination. Right mastoid, cavity of the radical mastoid operation, filled with pus of foul odor, post-auricular sinus with discharging pus of same type. Right nasal cavity shows signs of atrophy at the posterior part of the inferior turbinate and of the nasopharynx, both localities being covered by loose, white crusts of offensive odor. Wassermann negative.

Bacteriologic examination. Friedlaender bacillus and staphylococci.

Treatment with autogenous vaccine. Feb. 26, 1916, 50 million; March 1, 100 million; March 5, 200 million; March 9, 400 million; March 12, 800 million, general reaction; March 16, 1 billion; March 20, 1200 million, discharge from post-auricular sinus decreased, nasal condition unchanged. March 25, 1400 million; March 29, 1600 million; April 4, 1800 million, no change in the nose; April 9, 2 billion; April 15, 2200 million. No change in nasal condition. Treatment discontinued.

Case 23. M. R., female, 20 years old. *Family history* negative. *Past history:* Bad odor and crusts for many years.

Examination. Total atrophy of inferior and middle turbinates of the right side; partial atrophy of the left. Greenish, tenacious crusts of offensive odor covering the atrophic structures. Pharyngitis sicca. Wassermann negative.

Bacteriologic examination. Perez bacillus.

Treatment with autogenous vaccines. Oct. 28, 1916, 100 million; Nov. 3, 200 million; Nov. 7, 400 million; Nov. 11, 600 million; Nov. 16, 800 million, marked general reaction; Nov. 20, 1 billion, patient claims crusts to be loose and has to wash her nose twice instead of three times a day, but odor unchanged. Nov. 25, 1200 million; Nov. 30, 1400 million; Dec. 4, 1600 million; Dec. 9, 1800 million, crusts very loose and decreasing in quantity; Dec. 14, 2 bil-

lion; Dec. 19, 2200 million. Patient interrupted treatment until January 8, 1917. Meanwhile the crusts had again become tenacious and the patient had to wash his nose three times a day.

Jan. 8, 1917, 2400 million; Jan. 13, 2600 million; Jan. 17, 2800 million; Jan. 21, 3 billion. Crusts again looser, odor unchanged. Jan. 25, 3200 million; Jan. 30, 3400 million; Feb. 4, 3600 million. No further change. Patient washes nose twice a day. Crusts more easily removable but not decreased in quantity; odor unchanged. Treatment discontinued.

Case 24. J. G., male, 34 years old. *Family history* negative. *Past history:* For many years bad smell and crust formation, especially bad in cold weather. Patient washes his nose twice daily. No fondness for dogs.

Examination. Marked deviation of the septum to the left. Total atrophy of the right inferior and middle turbinates, both covered by dark green, thick crusts having foul odor. Wassermann negative.

Bacteriologic examination. Crust dropped into bouillon and incubated for 14 hours. A loopful of the broth culture was spread on four agar plates and incubated for 24 hours. Colonies fished into infusion agar slants. Examination of the growth on the slants revealed short, thick Gram negative bacillus with slight odor. When grown on ascitic broth the odor was more characteristic. Sugar reactions: *dextrose,* acid; *mannite,* no change; *lactose,* discoloration in gas tube, acid; *saccharose,* no change. Suggestive of Perez bacillus.

Animal experimentation. Rabbit No. 113 injected intravenously with 1 c. c. saline emulsion from agar slant on Oct. 19, 1916. Rabbit remained well; apparently no nasal changes.

Treatment with autogenous vaccines. Oct. 27, 1916, 100 million, severe local reaction; Nov. 5, 400 million; Nov. 9, 600 million. Patient claims that he feels better; odor slightly decreased, crusts looser. Nov. 14, 800 million; Nov. 18, 1 billion; Nov. 22, 1200 million. Patient washes his nose only once daily; odor unchanged. Nov. 26, 1400 million; Nov. 30, 1600 million; Dec. 5, 1800 million. No further change. Dec. 9, 2 billion; Dec. 15, 2200 million. Crusts again thicker; patient claims that the odor is worse than before. Dec. 19, 2400 million; Dec. 23, 2600 million. Treatment interrupted to Jan. 8, 1917. Crusts and odor unchanged. Jan. 8, 2800 million; Jan. 13, 3 billion; Jan. 17, 3200 million; Jan. 22, 3400 million. No further change. Treatment discontinued.

Summary of results obtained:

Case 1. Perez vaccine (Horn) negative. Autogenous vaccine, transient improvement with return of old condition.

Case 2. Hofer original vaccine negative. P., D. & Co. ozena vaccine (combined), transient improvement with return of old condition. Autogenous vaccine, negative.

Case 3. P., D. & Co. ozena vaccine (combined), condition of nose worse. Autogenous vaccine, no improvement.

Case 4. Frontal sinusitis.

Case 5. Frontal sinusitis and ethmoiditis.

Case 6. Autogenous vaccine negative.

Case 7. Lues.

Case 8. Lues.

Case 9. Horn's mixed vaccine negative.

Case 10. Frontal sinusitis.

Case 11. P., D. & Co. ozena vaccine (combined) negative.

Case 12. Autogenous vaccine negative.

Case 14. Mixed Perez vaccine (Horn), slight improvement in odor.

Case 15. P., D. & Co. ozena vaccine (combined), slight transient improvement in odor.

Case 16. P., D. & Co. ozena vaccine (combined) negative. Not conclusive as Wassermann was positive.

Case 17. Tuberculosis.

Case 18. Mixed Perez vaccine (Horn), improvement in odor, loosening of crusts. Was previously treated with autogenous vaccine with negative result.

Case 19. Original Hofer (Perez) vaccine negative. P., D. & Co. ozena vaccine (combined), negative. Autogenous vaccine, negative.

Case 20. Autogenous vaccine, negative. Mixed stock Perez vaccine (Horn), negative.

Case 21. Operative case.

Case 22. Autogenous vaccine, negative.

Case 23. Autogenous vaccine, crusts looser.

Case 24. Autogenous vaccine, negative.

The literature on the subject is quoted in the reports of Horn and Victors, before the American Medical Association, 1915, and before the American Laryngological, Rhinological and Biological Society, 1916.

1320 Madison Avenue.

APHASIA.

MRS. MAY KIRK SCRIPTURE, New York City.

The purpose of presenting a description of aphasia, especially in a work of this character, may not appear evident in view of the fact that it is, so far as we are aware, a loss of function due to cerebral lesion and hence outside the usual province of the teacher of speech. I feel warranted, however, in presenting the subject for several reasons. Most important is the fact that a study of the symptoms of "speechlessness" offers one of the best approaches to a comprehension of the nature and complexity of the speech mechanism in normal persons. Furthermore, the teacher of speech will inevitably come into contact with patients suffering from milder or partial aphasia and should be capable of making a diagnosis for his own purposes as well as taking a description of the symptoms that will be of assistance to the physician. And, contrary as it may seem, re-education is not without value even here.

I have spoken elsewhere of the value of localizing mental functions in the cerebrum. Nowhere has the study of the loss of skilled movements (apraxia) been pursued with greater zeal, nor indeed with more fruitful results, than in the field of language. In spite of the fact that progress in this direction has done so much to help us solve many of the intricate problems of psychology and pedagogy, we must use the facts obtained here with great caution for the evidence of localization of function is not yet complete, that is, for such specific localization as some authors in their zeal for this enticing theory have been led to claim. In studying the areas of the brain that have to do with the skilled movements of speech we are handicapped by the fact that the methods of extirpation and direct stimulation, which have been followed in locating functions analogous in animals and man, are not available here. Consequently the experiments which disease conducts for us furnish the largest part of our knowledge, and aphasia becomes vitally important. In summarizing the facts at hand I shall proceed from the grosser or the more general localizations to those of a more specific or minute nature.

One of the most remarkable and perhaps earliest discovered facts about brain localization is that all our higher mental life is controlled by one hemisphere; always that one which is opposite

the dextrous side of the body. It was also discovered in the early period of this research that there were large areas in the frontal portion of the cerebrum that apparently performed no function; they have been called the "silent areas." Cases have been cited to show that the connections between the hemispheres might be destroyed without the appearance of abnormality of function; and yet we cannot believe they are without purpose. In general we know that all the motor acts performed by the right hand are controlled from the left hemisphere, and contrariwise for the left hand. Forceful as such arguments are in pointing to specific functions for different brain centers, some of the phenomena together with the fact that not infrequently disturbances of function evidently due to lesion are suddenly restored, prepare us at the outset to expect no little amount of adaptability on the part of the nervous elements which compose the brain structure. Thus it becomes imperative that we approach the evidence for a more detailed localization with open mind.

The facts pointing to localization of speech functions are somewhat as follows:

Broca pointed out that the gyrus frontalis inferior of the left hemisphere was especially concerned in the power of speech. That this is the seat of "the faculty of articulate language" as he thought, is absurd. From similar anatomical evidence Wernicke assigned the sensory aspect of aphasia to the temporal lobe of the left hemisphere and made a distinction between "cortical," "subcortical" and "transcortical" aphasia. P. Marie states that the third frontal convolution of the left side has to do, if at all, only with the specifically motor parts of speech and that lesions here never result in a true aphasia; the difficulty resulting in aphasia lying beneath the Broca region, or in the neighborhood of the lenticular neucleus. When we include the opinion of some authors that aphasia never occurs without other mental disturbances though this opinion seems to be unsubstantiated, it is at once evident that localization of function on the anatomical side is a promising but unsettled question. At present it seems probable that Broca's region is intimately connected with the motor aspects of speech and that the rear part of the third frontal gyre has to do with the forming of sequence out of the elementary movements of speech; and it is more than likely that the whole frontal lobe is in one way or another involved in the production of speech. Indeed, it would appear that other and remote portions of the cerebrum are not without function in the production of verbal expression. From such a state of affairs it is evident that

we can entertain little hope of differentiating elements in the speech process on the basis of a purely anatomical localization of function. If we could say that *this* lobe of the brain has to do with innervating one part and *that*, another part, we should have a clear cut case against such a conception as a "speech faculty." But the difficulty is not inherent, rather it is one due to an absence of complete understanding of the neurological aspect of the problem.

It must be apparent that no chart of the brain can be drawn adequate to show the localization of all those parts involved in speech. However, I insert the one I use in my lectures which I find of great help in making the problem more concrete.

We come now to what from our point of view is a more fruitful line of investigation, the symptoms of aphasia. It is well to have an understanding of the term at the outset. Webster gives a good definition of the term for general purposes, when he defines it as "loss of power of speech or of the appropriate use of words, the vocal organs remaining intact, and the intelligence being preserved." This is, however, not a good working definition, for some cases well within the domain of aphasia, are not without impaired intelligence. Aphasics present the following symptoms:

1. An almost total absence of speech, retaining the use of one or two meaningless words or jargon.

2. A plentiful supply of words which he uses wrongly and without meaning (verbal amnesia and paraphasia).

3. He may be able to repeat a sentence, copy writing or print in his own hand writing, but be unable to give expression to his own thoughts. He may or may not understand the words to which he gives expression. (echolalia).

4. The patient may be able to carry out written or oral commands and still be unable to give expression to those commands.

On the side of perception (agnosia)

5. He may be able to understand what he hears and be able to write but unable to read, even what he has just written (alexia), or the reverse.

6. He may have lost the ability to recognize numerals and objects with which he is otherwise familiar (asymbolia). We may include here the inability to recognize colors (achromatopsia) to which he can nevertheless point when they are named; similarly he may be unable to recognize the size or shape of objects by handling them (astereognosis) when he is perfectly aware of their nature when presented in another way.

Not all of these difficulties, to be sure, properly fall in the category of aphasia, but they are very near akin and we have cited them as necessary to a correct interpretation of the facts.

The usual interpretation has been that each of these several functions were centrally dependant upon a particular group of images which, when they failed, resulted in such anomalies as those we have just given. It is supposed, let us say, that we have types of minds depending largely upon the types or relative efficiency of this or that kind of imagery the latter depending in turn upon the relative efficiency of the corresponding brain centers. Thus a person with a well developed auditory center will have a good memory for impressions received through the sense of hearing. His mind will be more receptive to that kind of impression and will recall auditory images with greater facility than a person whose auditory center is not so well developed. Such a person will learn best what he reads aloud or what is read aloud to him. Such a person is said to possess an audible type of mind.

A person whose visual center is especially well developed will be more readily receptive to impressions received through a sense of sight. Such a person remembers a thing best by seeing it. His reading consists in permitting his mind to get impressions of the words through the sense of sight. These word images are received by the brain and are stored up for further use. In recalling the word he merely calls up the image of the appearance of the word in print or in written form.

Still another type of mind is the motor type. This person's memory is best for muscular activity. Some authors go so far as to say that the part of all experiences retained are kinesthetic (Ribot: Le Vie Inconsciente et les Mouvments—Paris 1914); others that no thought is carried on without some sort of muscular innervation (Washburn, Movement and Mental Imagery). The theory is that this type of mind remembers in terms of muscular co-ordination and feelings. We are not so interested here in the theoretical aspect or correctness of the image theory as in its bearing on the problem of speech and the interpretation of the facts of aphasia. Are these facts to be interpreted as evidence of the absence of this or that kind of imagery or do they indicate that this theory is not correct?

We may distinguish, to begin with, between an exterior and an interior speech. Observation has led us to believe that in most cases of aphasia inner speech is intact, as demonstrated by the fact that the person can write; nor is his auditory imagery lacking for he can understand what is said to him. When in such circumstances

the patient cannot speak we must seek another explanation of the difficulty. And when he cannot write he may be able to read, which belies the idea that he is lacking in visual imagery. Or he may be able to read or repeat words when they have no meaning for him. In the majority of the cases of aphasia the imagery is not absent. The source of the difficulty here is the absence of the ability to order the imagery, that is, voluntarily, to result in external speech. The fact that he can understand is explained when it is remembered that in reading or listening to another this sequence is ordered for him. On the receptive side, agnosia, the fault is not with the imagery for the object is most often directly presented to the senses, the fault here is due to a failure of those processes that lie behind the act of perception. In these two cases the patient's thinking is unimpaired; but there is a form of aphasia in which the thought is impaired, where the thought symbols, images, or whatever they are, are lacking and as a result the person can neither grasp nor retain a new proposition.

Before leaving the problem of imagery I must call your attention to several almost self-evident truths. The congenitally deaf person does not learn to speak of his own initiative; the importance of auditory cues is most evident. On the same basis the congenitally blind do not learn to write. But neither seems to be hampered in their thinking. On the other hand the vividness of a particular image does not insure at all the amount of actual knowledge that attends it. You may have a most vivid image of some house and be unable to tell how many windows there are in it, or a most vivid image of a melody and not be able to play it without the music before you. There is no doubt but that we differ in the kind of imagery we use. It is not infrequent that we think we think in auditory imagery when talking, in kinesthetic imagery when writing, and perhaps quite universally in visual imagery when dreaming, but these seem rather habitual than fundamental to the thought process. Now in external speech, as we noted above, auditory cues seem to be necessary; and in writing visual cues seem to be necessary but it does not follow that a failure in these functions is due to the absence of imagery, but to the inability to use or voluntarily order these processes. The same argument holds for imperception; we may retain all our imagery and still be unable to interpret the facts presented to us. And finally the patient may have lost the ability to frame, even within his own mind, consecutive thoughts, may have been deprived of any inner speech and yet may be able to think and comprehend, though in an inferior manner.

What then is the teaching of aphasia as regards the process of speech? Some expressions of a low character, usually of an emotional character, may be produced reflexly as shown by the meaningless or jargon expressions of one type of aphasia. It is also evident that the patient can formulate what he wishes to say in his mind, he can write it, and yet be unable to express it orally which indicates that external speech whether expressed in subtle movements of the larynx, etc., or aloud is not identical with the formulation of our thoughts for our own conception. The fact that he can comprehend an expression which he cannot formulate indicates that these two processes are not identical and that back of the latter, back of all spontaneous speech there is an element of volition. Imperception demonstrates that there is a process behind the internal speech, the flow of imagery, with results in their having significance. We find thus three steps in the language process: 1. The knowing of what is to be said. 2. The formulation of this in terms of imagery or internal speech. 3. The complicated process of innervation which results in the actual expression.

As regards an analysis of aphasia, it is evident that a classification based upon loss of imagery is necessarily imperfect. The first distinction to be made is between motor and sensory aphasia. The former can always be distinguished from the latter by the patient's ability to write. Motor aphasia may present an almost total absence of words or a copious vocabulary incorrectly used. Sensory aphasia, apart from the varieties we have already noted, may present a situation where the patient is able to comprehend ideas already formulated for him but which he cannot voluntarily construct or he may be able to present ideas which he fails to comprehend when they are presented to him. A rule which has held good in all my experiences is that the more complex and voluntary parts of speech suffer first, the more automatic being the last affected.

The type of a lesion that may cause an aphasia is usually circulatory, and, therefore, apoplectic. It is only rarely due to the presence of a new growth, to abscess or to softening. This probably accounts for the fact that attacks of aphasia are usually sudden in appearance and many times are only of short duration. Among the causes of aphasia may be included, excessive drinking of alcoholic liquors, excess of smoking, severe heat or cold, etc. We have known of cases where a great fear or anger has induced aphasia. The most potent causes are, of course, organic in nature, the condition often resulting from a state of low vitality due to severe nervous stress or overwork.

While the teacher of speech will, as a rule, not be competent to deal with aphasia there is much he can do to aid in recovery. Every effort should be made to bring about a general condition of bodily health and vigor. When that is accomplished the patient must be induced to make an effort to recover his speech. Not infrequently it happens that the unhappy condition will disappear as quickly and as completely as it set in.

EXAMINATION FOR APHASIA.

1. General Condition
2. Organs of Enunciation
3. Activity of Senses
4. Understanding of Sensations
   Does a patient jerk away from light?
   Does a patient lean toward sound?
   Does a patient turn head and eyes toward sound?
   Does a patient look toward place where you touch him?
5. Understanding of Objects
   Does a patient understand objects shown to him in any way? (cut with knife, prick with pin, hat, glass, water?)
6. Understanding of Movements
   Does a patient understand movements? threatening? eating?
7. Understanding of Signals
   Bell, whistle, flag?
8. Understandigg of Speech
   Can he point to objects named? Can he execute commands he hears?
   Agreement of object seen with name heard?
   Object touched with name heard?
9. Understanding of Writing
   Point to objects whose names he sees? Execute commands read?
   Agreement of printed word with heard word?
10. Spontaneous Speech
    What does patient say of his own accord?
    Can he answer questions?
    Is his syntax correct?
    Automatic series (1, 2, 3,—— Lord's Prayer).
11. Spontaneous Writing
    Can he write answers to questions? His name? His address?
12. Repeating Speech
    Can he repeat what he hears? Words? Short sentences? Long sentences?
13. Dictation
    Can he write from dictation? Short sentences? Long sentences?
14. Copying
    Can he copy what he reads in print?
15. Reading Aloud
    Can he read single letters? words? sentences?
16. List of Commands for Patient to Read and Execute
    Put on your hat. Put out your tongue. Shut your eyes. Show your teech. Raise your hand. Smile. Laugh. Shake hands. Put your hand in your pocket.

Aphonia. The term used in medicine is meant to signify a more or less complete loss of voice. It is distinct from mutism, in which it is impossible to form articulate sounds, and in most cases the voice is not entirely lost. The voice is essentially produced by

three agents: viz. 1. the expiration or air, 2. the opening of the glottis, and 3. the tension of the vocal cords; and hence anything interfering with expiration or with the function of the glottis or the vocal cords, may cause aphonia. Thus, it may result from paralysis of the respiratory muscles, or from pneumonia; or it may be caused by diseases of the larynx, as chronic laryngitis, edema of the glottis, polypus, etc.; or by pressure upon the larynx by abscesses, vegetations, and any kind of morbid growths; or it may be traced to some functional or organic disturbance of the vocal cords. The muscular fibres which act upon these cords may become affected in acute laryngitis by extension of the inflammation, or their action may become impeded by the pressure of false membrane in croup. Aphonia may often be traced to compression of the recurrent or inferior laryngeal nerve, which is the nerve supplying motor power to all the larynx except one. Such pressure is not infrequently caused by aneurism, abscess, or tumor. A wound or contusion of the pneumogastric nerve, or one of the recurrent branches, will cause aphonia or, more commonly, extreme hoarseness from paralysis of the laryngeal muscles on one side. Aphonia is moreover very commonly associated with hysteria.

The causes may be hysteria, paralysis of the vocal cords, paralysis of the muscles of the larynx, tumor of the larynx.

55 Central Park West.

Pituitary Tumor—Operation by Transsphenoidal Route. F. O. Lewis, *Pennsylvania Med. Jour.*, Feb., 1918.

The tumor was an endothelioma and gave rise to the following symptoms: Headache, hemianopsia, somnolence and adiposity. About a year previously the patient had had an accident, falling down a flight of stairs and landing on the back of his head. He was stunned but not unconscious. Severe headache since the time of the fall—occipital at first, but later more severe over the frontal region. Roentgen examination showed almost complete destruction by pressure of the entire floor of the middle fossa—sella turcica, sphenoid sinus and body of sphenoid entirely destroyed. The transsphenoidal operation was done under intratracheal anesthesia, but the outcome was fatal. Ed.

DEFECTIVE SPEECH AND SOME OF ITS PHASES.*

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The subject of defective speech covers such a wide scope of the human anatomy and pathological physiology, that I thought it best to limit myself to a few of the most prevalent conditions. Among all the ills and miseries that befalls humanity, there are none so embarrassing and entail so much anguish and depression as that of defective speech. Dr. Hudson Makuen, the pioneer of defective speech in this country, had the courage to call attention of the profession to its indifference, and through the media of the profession, to spread a propaganda of the importance of defective speech. For some time the profession seemed to be indifferent and the laity in general showed total negligence if not distinct levity. All cases that come into our clinic practically have their own sad tales. Some children left school to avoid embarrassment during recitations, and to eliminate laughter and scorn in the class rooms. Many men with good brains have lost their positions, and some could not obtain the positions that they deserve on account of defective speech.

To illustrate. We now have a little girl in our clinic, age nine, somewhat backward, probably of a lowered mentality than the average child of her age, who becomes embarrassed upon the slightest provocation especially when among strangers. The children at school, not understanding her weakness, nor the teachers understanding her sensitiveness, are constantly teasing, nagging and making life miserable for her. The parents at home also consider her a back number. Their general attitude is that Catherine cannot do this, and Catherine cannot do that. On account of constant oppression and unfit surroundings, the child developed the typical physical carriage of that of a miserable individual, her head constantly lowered, a face of anxiety, and when spoken to, her eyes are constantly directed towards the floor, and practically always has the answer "I can't." For the past few weeks, since the home surroundings have been bettered, due to a confidential talk with the parents, instructing them to be more tolerant with her, and the sympathy shown her in the Speech Clinic at the Polyclinic Hospital, the change in her general attitude is remarkable.

In these days, when efficiency is an important factor for substantial existence, it is our duty as healers of mankind to alleviate

*Read before the Philadelphia Laryngological Society, Dec. 4, 1917.

all misery, and to promote human welfare. Defective eyes or flat feet and many such ailments that are not acutely dangerous to human life, are treated with caution, and remedial defects are immediately instituted; but when incipient defective speech develops, we all have the tendency to say that it is neurasthenic, and in children the old proverb is constantly repeated that they will grow out of it. The more I see of defective speech, the more I am convinced that they grow into it, instead of out of it, and it is therefore essential that speech be corrected early before it becomes chronic.

The first form of defective speech to be considered is dyslalia or stammering. There are three stages in the stammerer's course: (1) Acute, which is characterized by rapid gabbling speech, confusion of thought, irritability, and difficulty in concentration; (2) The above symptoms exaggerated with a tendency to stammer, etc.; (3) continued stammering.

The most can be accomplished in the early stage, if proper environment is instituted. Great caution should be taken not to irritate the child, nor to punish it during the early stage of excitement, as corporal punishment will produce a vicious cycle and exaggerate the above symptoms.

Speech is an acquired faculty, and defective speech is a faulty faculty acquired. It is therefore essential to institute treatment before faulty speech is developed, for in treatment it is necessary to undo all the faults and to begin anew to teach proper methods.

(3) To illustrate a case of second stage of stammering. Master B. Age 14. Weighs 128 lbs., 5 feet 5 inches, left school in seventh B. grade and is the oldest of a family of six; his habits are good, and he is a constant reader. About five months ago, he was compelled to stop school on account of financial conditions, to seek employment. Four months ago, he became very irritable, developed a temper upon the slightest provocation, which was totally neglected by the parents. Five weeks ago, he noticed that he occasionally stammers, especially in public. His parents are taking keen interest in him, and willing to deprive themselves of his little income so as to put him in the right hands and on the road to complete recovery.

The essential basic principles for speech are breath, voice, articulation and mentality.

(1) The breath is attained by the lungs, diaphragm and the accessory respiratory muscles. In speech we need very little air, but it is important to teach the defective speaker to control the air

column, so that there is no sudden outburst following full inspiration nor the gasping following the complete expiration.

(2) The late Dr. Hudson Makuen defined that voice is produced by a column of air, playing over the vocal cords setting them into vibration, and its diffusion through the various remote resonate chambers and into the atmosphere. Voice is an early development and precedes that of speech, for the baby whines before it speaks, and as mentality develops the desire for expression arises.

(3) At first various sounds are uttered and gradually the sounds are symbolically clothed in the form of words, and when properly thought and articulated, normal speech is the result. The essential things for proper spontaneous speaking therefore are: (a) Ideas, which are stimulated by the occasion the person is confronted with. (b) The expression and the external realization of these thoughts either in the form of writing or speaking. The latter is probably accomplished by central innervation, and through efferent innervations conducted to the peripheral organs of speech. The primary principles of speech are, the auditory and glosso-kinetic centers, the visual and chiro-kinetic center, the latter being chiefly employed in writing. In the auditory centers are stored the images of the words as they are spoken and heard, while in the glosso-kinetic centers are stored various memories of complicated muscle movements and modes of co-ordination of speech. Ideas arise and are interpreted in the form of words stored in the auditory center and are transmitted to the peripheral muscles of speech by the glosso-kinetic centers.

The phenomena of stammering are usually characterized by a contraction of the muscles of speech with the resultant movement of other related organs. The latter is due to misdirected concentrated uncontrolled nerve energy which, during the stage of excitement, is disseminated to the neighboring organs sometimes resembling that of chorea; while the articulatory defective speakers, unless due to some mechanical reasons such as elongated frenum, hypertrophied tonsils and adenoids are due to faulty auditory perception and faulty glosso-kinetic emanation. Under this heading would come the so-called cute or baby talk, where the *D* is pronounced as *DZ*, doll such as dzoll, cat as tat, etc., and many other such faulty articulations. Many parents encourage the children afflicted with this so-called cute or baby doll disease, on account of its oddness and cuteness to the listener.

Speech is normally automatic and independent of conscious effort. This is true whether speech be normal or abnormal. De-

fective speech is therefore auto-incurable unless the conscientiousness of the individual is aroused and proper interferenc is instituted; while voice is an art acquired and is often the mirror of man's own self, for we are all acquainted with the husky voice of the vagabond, the tremulous voice of the asthenic and overworked, the sympathetic voice of the physician, the cold blooded voice of the lawyer, the chest tone of the factory girl, the pious voice of the clergyman, and the falsetto voice of the feminized masculine.

Thus far, I briefly touched upon the stammerers and the so-called baby talkers. Among the other phases of defective speech are: the husky or hoarse voice, which may be due to acute and chronic laryngitis, tuberculous, syphilitic laryngitis, intra-laryngeal tumors, papilloma, and tumors pressing upon the recurrent laryngeal nerve, etc. Whispered voice, which is often followed by complete aphonia, may present organic bases such as complete or partial paresis or paralysis of the adductor muscles of the larynx, while others are of the so-called hysterical type. In normal whispering there is a triangular shaped opening in the glottis between the projecting parts of the arytenoids. There may, however, be individual adductor paralysis, for when the inter arytenoids are involved, the same phenomena results, for in the latter condition there is faulty approximation of the arytenoids. When the thyroarytenoidei muscle is at fault, there is an eliptical slit widest at the middle of the vocal cords, due to faulty support of them. When there is crico thyroid weakness which normally stretches the cords, there will be a sinus aspect, while weakness of the crico arytenoid lateralis results in faulty approximation of the artyenoids. Here I wish to quote two cases brought by their physicians to the Pennsylvania Hospital for a direct laryngoscopic examination.

Case No. 1. Mrs. K., Age 57, married, has family of four children, of moderate means, who five months prior to my examination contracted acute laryngitis. The physician properly instructed her to speak as little as possible and if necessary to speak, she must whisper. Among the other treatments instituted were direct intralaryngeal applications of argyrol, glycerite of tannin, and various inhalations, etc. The larynx was examined by the direct and indirect methods, and I could find no organic trouble, but I noticed that when the patient whispered, there was an undue column of air expirated. This was definitely proven to myself and shown to the patient that by placing a mirror in front of her mouth a thick film of moisture settled upon the mirror; while the same sentences uttered by myself barely clouded the mirror.

Case 2. Mrs. B., married, has family of three children, hoarse for a year.

Direct and indirect laryngoscopic examinations negative. This patient practically went through the same treatment without any improvement. The above mirror observations held true here also. I found that these patients had no diaphragmatic control and practically spoke on the residual pulmonary air. After about ten lessons of proper diaphragmatic control, and proper vocalization, we were able to produce normal tones, which was followed by complete recovery. The only other mechanical means used with these patients was asking them to cough at various intervals, for thus I brought out voice during their coughing spells, and called their attention to it. I also used pressure on each side of the thyroid cartilage pressing inwardly from both sides, thus aiding the muscles that have been relaxed for such a long time.

Falsetto voice. Two cases came under my observation, one a school teacher, female, age 23, who after several months of teaching developed a high pitched falsetto voice. Her only dread was, of the many remarks made, that she was developing the typical old maid's voice. Intralaryngeal examination was negative. The difficulty that this young woman was confronted with, was, that she taught in a truant school, and in order to accomplish the duties intrusted to her, it required a great deal of loud talking on her part. The larynx seemed to be in a higher place than normal, which, therefore, threw the sound waves higher with the resultant falsetto voice. Careful vocalization, laying great stress upon the respiratory control, and constant exercising of the low and middle register brought the voice back to its normal state. This condition occasionally recurs during excitement.

The second case was a boy, age 19, brought from Wilmington by his sister with a note from the family physician for a tonsillectomy. Upon examination I found two innocent tonsils; intralaryngeal examination negative. I also examined to see whether there was any sexual defect, but this was not present. The falsetto voice developed between the ages of 14 and 15, when there was a break in the voice, between that of his natural voice and the falsetto. He found that it was much easier for him to speak in the falsetto and he adopted the latter. After many lessons in vocalization, and suitable exercises upon the low register, I firmly established a reasonant basso voice. The difficulty I had with him was that he had no musical ear and could not imitate tones.

Muffled voice, which is characterized by its dullness, is caused by nasal and naso-pharyngeal obstructions. This can readily be cured by operative interference, such as tonsil and adenoid removal, and selective operation for nasal obstruction.

Nasal voice is caused by inadequacy of the velum palati and levator palati muscles allowing a great proportion of vibrating air to escape through the naso-pharynx. This condition often accompanies atrophic rhinitis, and injury to the soft palate and tonsillar pillars following poor tonsil surgery.

Summary. (1) It is important that each patient be studied from various angles, that the ear, nose and throat, eyes, medical and neurological examination be made, as it is necessary to seek any etiological factors and eliminate them. (2) Surgical interference should be instituted when necessary, but one must not be left with the impression that the latter will cure all forms of defective speech. (3) It is essential that the eyes and ears be trained by constant repetition of various exercises, first that they be seen perfectly and be pronounced properly. We know from experince that the eye can be trained to within a thousandth of an inch. Taking the eye as a simile, it is perfectly logical that the ear could also be trained. As previously stated, words are stored in the auditory center and transmitted through the glosso-kinetic center. Therefore, by constant repetition of exercises and words, the auditory center is impressed with the true sound of the various words, which can readily be recalled and properly be expressed when the occasion presents itself. (4) Speech is normally automatic and subjectively independent of conscious effort. A person afflicted with defective speech, realizes when others speak faulty or mis-pronounce words, but are unable to correct themselves. In order to accomplish most in the least time, it is essential that treatment be instituted early before it becomes chronic. (5) Defective speech is an overwhelming problem confronting us in these days of war and triumph, for there are innumerable stammerers in the European armies now, and thousands who become deaf. As otologists and laryngologists, we will be confronted with this immense problem and must, therefore, be prepared to teach those who stammer, and to teach lip reading to those unfortunate deaf who will return from the battlefields.

1736 Pine Street.

SOCIETY PROCEEDINGS.

NEW YORK ACADEMY OF MEDICINE.

SECTION ON LARYNGOLOGY AND RHINOLOGY.

November 28, 1917.

Status Lymphaticus; Death Following Tonsillectomy; Autopsy. DR. WILLIAM WESLEY CARTER.

On Oct. 4 there was, among the fourteen cases of adenoids and tonsils to be operated upon that afternoon in my service at Gouverneur Hospital, a little girl five years of age. The child was of a nervous and excitable temperament and was terribly frightened on being admitted to the hospital. She was poorly nourished, but there was no evidence of any definite organic or constitutional abnormality. No family or previous personal history could be elicited bearing upon the condition from which she succumbed.

The tonsils were enlarged and were of the submerged variety. Adenoids were in moderate amount.

At 2 P. M. this child was etherized by an expert anesthetist. The tonsils were enucleated by the Sluder method, and the adenoids were removed with the Gottstin curette. The actual operating time was not over two minutes. There was only moderate bleeding and the patient was removed to the ward in good condition. At 5:55 P. M., three hours after the operation, and after the child had recovered from the anesthetic, the nurse noticed that she suddenly became cyanosed. Death ensued before the house surgeon could be summoned. The usual means were employed to revive the patient.

The autopsy was performed by Dr. Schwartz three hours after death. The following were the salient features noted in the report:

Thymus gland enlarged, reaching over base of heart and blood vessels. Weight 21½ grams. No constriction of the trachea was noted.

Heart: Foramen ovale open. Valves normal.

Larynx, trachea and bronchi normal. No evidence of post-operative hemorrhage.

Spleen normal in size. Malphigian corpuscles unusually prominent.

Mesenteric glands and solitary lymph follicles of the intestine enlarged. Retro-peritoneal glands enormously enlarged, these being the size of hazel nuts.

Kidneys normal.

Pathological diagnosis: Status lymphaticus.

I am prompted to report this case because of the rarity of this disease, because of the uncertainty which surrounds its pathology, because of its grave significance in operative cases, and because the profession should be constantly reminded that there is such a condition as status lymphaticus and that it is a menace which must be reckoned with by every operator. It is a danger too which in the present state of our knowledge, cannot be forestalled and guarded against.

These cases are not common. In my own experience which includes many thousands of adenoid and tonsil operations, and a large number of other conditions requiring operation under a general anesthetic, I have met with only two cases of status lymphaticus, both of which were first identified at autopsy.

According to Bovaird and Nicoll who weighed and measured the thymus in 495 consecutive autopsies, this gland is 3 cm. wide and weighs 7 gms. in a child one year old. It increases in size for three or four years and

then gradually decreases until puberty is reached, when it either disappears or remains throughout life in a much atrophied condition.

The anatomical relations of the thymus are important. A study of these will indicate at once how an enlarged gland may press upon adjacent vital structures; this is especially true since the thymus lies between two unyielding bony structures, the vertebral column and the sternum, the distance between these up to the second year being only 2 cm. The phrenic nerves and the pericardiac phrenic artery are in relation with the thymus on both sides. The lower pole of the gland rests upon the pericardium, reaching usually to the fifth costal cartilage; the posterior surface lying upon the right auricle and ascending aorta and sometimes reaching as far as the bifurcation of the trachea. On the right side it is in contact with the superior vena cava and right innominate vein. The gland has a rich arterial supply, but the veins emerging from it constitute only a single small plexus. This disproportion between the afferent and efferent blood vessels would seem to favor rapid engorgement of the gland. This has been claimed by some writers to be the cause of sudden death following fits of anger or prolonged crying spells.

The various conditions caused by enlargement of the gland have been stated by Pfaundler to be: (1) Tracheal stenosis. (2) Conditions caused by pressure by the gland upon the circulatory system and nerves. (3) Status lymphaticus. (4) Laryngo-spasm. (5) Discrasias caused by functional disturbances of the gland itself.

Only two views have received wide acceptance: (1) That there is a neurosis of some sort, toxic or otherwise and that death is a cardiac death. (2) That there is a compression of the trachea and that death is due to strangulation.

Apparently the case which I report to-night was a cardiac death. These are the cases that give no warning and the only evidence is revealed at the autopsy.

In operative cases death may occur during the administration of the anesthetic, two or three hours, or as long as twenty-four hours after the operation. In my opinion the anesthetic and not shock from the operation is usually the important factor in determining the fatal issue in thymus cases.

In regard to compression of the trachea and death by strangulation: These cases as a rule give a history of gradually increasing dyspnoea extending over a period of weeks or months. In 1904 Jackson reported the case of a child 4 years of age in which the compression was demonstrated by the aid of the bronchoscope. The dyspnoea was relieved by using a long tracheotomy tube and cure was later effected by removing the thymus.

There have also been reported a number of cases of impending strangulation that have been relieved by the surgical removal of a portion of the gland, or by pulling the gland forward and stitching it to the sternum.

One case is reported as having been relieved by X-ray treatment.

In 1904 I reported before this Section a case of thymic tracheo-stenosis. The patient, a baby five weeks old, was brought to the hospital suffering from extreme dyspnoea. This condition had existed from birth and had been getting much worse. Tracheotomy and the introduction of a long tube relieved the dyspnoea, but the child died later from bronchopneumonia.

There is at present, so far as I know, no certain means for diagnosing this condition in children. Probably the X-ray is best, but even its evidence is fallacious and at best can scarcely be considered more than corroborative.

A closer study and a more intimate knowledge of the internal secretion of the thymus gland may suggest some test either of the blood or the secretions which may enable us to unravel the mystery which now surrounds the pathology of this gland.

DISCUSSION.

DR. MAYER said that the section was indebted to Dr. Carter for bringing this matter before the members for it was an accident that might happen to anyone. He then told of a child of ten whom he had operated upon for the removal of Tonsils and Adenoids quite a number of years ago. The child was stout with a pasty complexion and did not seem to the speaker to be able to throw off any complication that might arise, and he therefore surrounded the patient with every precaution, insisting that the patient must remain in the hospital for at least a week and the anesthetic given was reduced in amount to the minimum. In spite of this care it took that boy three days to become fully awake. He acted as though he were profoundly narcotized after a dose of scopolamine or morphia and the speaker believed that if unusual care had not been taken the patient would have died, and he always looked upon it as one belonging to the category which the speaker had mentioned. Not every child is capable of successful operation as such cases as are under discussion clearly show.

DR. MACKENTY said that he believed status lymphaticus to be very common, but that fortunately we do not often see deaths from it. Many cases on the borderline often escape death. He had had two deaths, both in children, one of them six to eight months old. This child died suddenly, six hours after an operation. The other was two years old, with very peculiar tonsils—large, pale, and boggy looking. One month ago he saw a child with the same kind of tonsils, and felt worried about the case. Suddenly, while he was watching it, hours after operation, the child stopped breathing, and appeared to be dead. However, he managed to restore breathing. This was probably a borderline case.

DR. FREUDENTHAL said that these cases are very rare. He also had operated on many thousands of tonsils, and only two years ago he met with the same accident as that reported by Dr. Carter. The child was a strong boy about ten years of age, who was operated in the usual way and then removed to the ward, no unusual symptoms being visible. An hour later, the house surgeon saw the child, and he was sitting up. A little later, when the house surgeon was not at hand, the child suddenly died. Dr. Freudenthal said he had met very few such cases, but they do occur. It is time that the public was enlightened to the fact that an operation on the tonsils is really an operation, and that accidents may and do happen. Not only ought clinical patients to be taught this, but others as well.

DR CARTER said there is no way of proving how frequently status lymphaticus occurs, for we only hear of those cases that result fatally. Another question he would like to have answered was whether this could be called a pathological condition or would it be called a disease. He had purposely called it both in his presentation of the subject, hoping to have this criticized. It is characterized by the enlargement of the lymphatic glands of the thymus—a definite pathological condition. He had once called it a disease and the accuracy of the term had been questioned.

Of course, the report of such cases as this is not a good business proposition, for if people should know that such an accident as this might occur and that it cannot be forestalled, the number of tonsil and adenoid operations might be materially reduced, but we should not consider that question.

Two Cases Representing End Results After Laryngectomy. DR. D. B. DELAVAN.

Having been misquoted as saying that all cases of malignant disease of the throat die, Dr. Delavan presented a patient to prove that under prompt and skillful treatment, some, at least, may be saved. The first patient presented was operated upon by Dr. B. Farquhar Curtis in 1898, nearly twenty years ago. About two-thirds of the larynx was removed. Microscopical examination proved the growth to be epithelioma. Since

the operation, there has never been any sign of recurrence. The voice has been good, and the patient has engaged actively and successfully in the arduous occupation of traveling salesman.

The second case was that of a young lady under thirty, whose entire larynx was removed six months ago by Dr. H. H. Janeway of the Memorial Hospital, N. Y., for an extensive laryngeal epithelioma. Her condition is as favorable as can be expected after so serious an operation.

These two cases were presented because of the strong contrast which they illustrate. The successful result in the case of the man had been due in no small degree to the calmness, patience, and courageousness of his temperament, considerations not to be ignored in the preliminary prognosis.

Dr. Delavan believes: (1) that life may be prolonged by surgical interference in favorable cases operated upon early in the development of the disease.

(2) In estimating what cases are favorable for operation, the character and temperament of the patient are factors of great importance.

(3) The reports of cases made soon after the time of operation give no further information of value as to the ultimate success of the work. It is only the end results that can show the real success after operation. Unfortunately, these are seldom published excepting when the result has been unusually fortunate. The fact that a patient has successfully survived operation is only the beginning of the story. The natural questions are: (1) How long was life prolonged after operation? (2) What was the condition of the patient meanwhile?

The man presented was a good example of the best results. How many of those operated upon have done as well?

Skillern's Operation for Empyema of Antrum of Highmore. DR. T. J. HARRIS.

Dr. Harris said that there are many operations for the radical operation and drainage of the antrum of Highmore. In recent years a great deal of emphasis has been laid upon the intra-nasal operation, and most men are acquainted with the so-called Canfield operation. Two or three years ago Dr. Skillern suggested a modification of the Canfield operation, with the idea of avoiding the removing of the inferior turbinate and thus doing a less extensive operation.

Four weeks ago he had performed the Skillern operation on the patient presented, with the aid of local anesthesia, and the operation was completely successful so far as the exposure of the antrum, complete drainage, and cure were concerned. Speaking from one case only, however, and with the possibility of faulty technique in mind, he encountered several objections to the method. With the greatest care, he was unable to secure complete anesthesia after repeated attempts. The young lady was an excellent patient, but she flinched under the chisel. Second, the bone was exceedingly thick. Without the inferior turbinate being removed at all, the bone was removed in the aperture pyreformic on the left side. The bone was exceedingly thick, and it was impossible to do this operation easily. Dr. Skillern says that it is difficult to keep the opening free, and those who had examined this case had probably noted that the opening is now nearly closed. As long as it was kept packed, it stayed open. Dr. Harris said that he thoroughly believed in the advisability of maintaining a permanent opening.

DISCUSSION.

DR. VOISLAWSKY said that it has been his privilege several times to see Dr. Skillern perform this operation, and he had also seen four or five cases operated upon by him. Dr. Skillern uses a burr, having several sizes, and there is no shock at all with this method. The great advantage of the operation is that it gives a very good view of the inside of the antrum. If there are any diseased parts they can be reached with any desired medication through this opening. Dr. Skillern curetted diseased

and polypoid tissue and removed small polyps through the opening that he made. Dr. Skillern also found difficulty in keeping it open, but that is not strange, for it is difficult to keep open any wound of the antrum. After a week or ten days of proper treatment through such an opening, it would seem wise to allow the opening to close of its own accord. After the removal of all polypi or granulations, if there were any necessity of doing further washing, the operation could be supplemented by washing, as has always been done, from beneath the interior turbinate.

Dr. Coffin said that he had seen Dr. Harris's case while the opening was still patent, and it did give a very good view of the antrum, and, as Dr. Voislawsky had said, one could treat the greater part of the interior or reach it by instrumentation. To Dr. Coffin it seemed that it was a very fortunate provision of nature that the opening does close up. He had been about to make the same suggestion as Dr. Voislawsky, i. e., that the advantage of the opening was the possibility of treating the antrum directly as long as necessary, and making subsequent drainage into the inferior meatus. It brought to mind what Killian had said—that in operating an antrum intranasally it was his effort to take out the middle portion of the inferior turbinate and open the antrum far back, as many persons whose antrum is open anteriorly complain of the dripping. At the time Dr. Coffin saw Dr. Harris's case, he had thought that if such an opening were permanent, he should be sorry to have such an operation done on himself.

Inquiring whether the operation was done under local anesthesia, he was answered in the affirmative.

Dr. Harris, in closing the discussion, said that it was only by occasionally seeing other methods of operation that we are able to determine whether our own are antiquated. One operation is not sufficient to determine its value, and surely Dr. Skillern would not recommend it if he did not feel that it was a worth while operation. There was no burr at the hospital where this operation was performed, and as Dr. Skillern says use a burr or chisel, in this instance he had to depend on the chisel. Personally, he found the operation a tedious one. The bone was very thick, and as compared with the quick work that he has been accustomed to, it was a long operation. The thought of the opening needing to be only temporary was a new thought to him. He had always felt very comfortable in having a permanent opening from the nose into the antrum, and had always felt that in that way he could take care of any subsequent infection.

Dr. J. L. Maybaum referred to two cases of chronic empyema of the maxillary sinus upon whom he had done the Skillern operation. In one, partial success was obtained as far as the patency of the opening was concerned; the opening closed at the expiration of six weeks at which time the discharge had entirely ceased. The second patient was a woman of 58 upon whom at first he hesitated to operate, but finally consented because of symptoms of focal infection. The opening has remained patent for fully six months; this result he ascribes to the careful packing for a period of two weeks, changed every other day, and to the flap of mucous membrane which he allowed to fall into the antrum. After discontinuance of packing, the patient was allowed to irrigate her antrum daily. It was perfectly possible to inspect the different walls of the antrum and make direct applications of argyrol, silver-nitrate solution, etc.

He had found no difficulty in obtaining good anesthesia in the case which still remained open by employing deep injections of novacaine 1 per cent sol. under the muco-periosteum of the facial wall, as well as into the lateral wall. A chisel was used to break down the crista pyriformis. In this case the lining mucous membrane had undergone marked polypoid degeneration so that no anesthesia was required for curetting it. Curetting of first case was somewhat painful, but this can be obviated by packing with 10 per cent cocaine-adrenalin gauze after opening the antrum.

Dr. Voislawsky and his colleague, Dr. Westley M. Hunt, have done such operations in the office, using a chisel, but they had given it up,

in as much as the chiseling seemed too much of a shock and they did not have the necessary burr. In ten days after operation the wound was ready to close up, and it was allowed to do so. Dr. Hunt washed the antrum a number of times through the inferior turbinate route.

Angioma of the Soft Palate. DR. T. J. HARRIS.

Dr. Harris said that this case was so unique in his own experience that he thought it would be interesting to the Section. The young man was eighteen years old, and for a year or more has had a swelling over the cheek. Seven months ago he developed a swelling in the mouth, but only in the last few weeks had he experienced any difficulty in swallowing. There was no pain. When the case was first seen, it was thought to be a cyst, but Dr. Coffin, who had seen a somewhat similar case, made a diagnosis of angioma. This was confirmed by aspiration, which yielded pure blood. The interesting questions were: What is the source of the growth and what is the operative procedure?

DISCUSSION.

DR. CARTER said that as he understood it, the development of this tumor dated back about a year and was not ante-dated by any traumatism. It would seem that a tumor of this kind, developing within a year and presenting the appearance that it would more clearly coincide with the history and appearance of a sarcoma, and he was inclined to regard it as such. He did not think that the aspiration of blood from the tumor would preclude that diagnosis, for the sarcomata, especially the round-cell ones, are very vascular indeed, and the fluid aspirated from them would be more than apt to be blood.

DR. ARROWSMITH said that Dr. Carter had expressed his views.

DR. TALBOT R. CHAMBERS said that he had a similar case upon which he had operated, several years ago. He thought then that it was a cyst from the gland. The patient had recently returned to the clinic with recurrence. Incision and pressure employed. But the swelling will probably recur. At the time of operation, nothing was evacuated excepting a bloody fluid; but he had not thought of its being an angioma. He regarded it as being merely a broken down cystic condition. He would like to know on what the diagnosis was based. He promised to bring the case to the next meeting of the Section.

DR. COFFIN said that he had seen the case previously and had also seen several others of like appearance. One of them had been presented before the Section several years ago. In that case Dr. Jonathan Wright was consulted as to the condition and had expressed himself as believing it to be angiomatous. This tumor pulsates both inside and out. It did seem a peculiar development for the length of time. Through Dr. Harris' courtesy he had presented the case the preceding evening, before a medical society in which were several well known surgeons who agreed with the diagnosis of an angiomatous tumor. Dr. Hirsch was there and stated that he had obtained very good results in such growths elsewhere on the body by the application of the x-ray, and he thought this case presented a favorable opportunity for such treatment; he stated that much larger doses could be used over the wet mucous membrane than over growths covered by skin. Whenever Dr. Harris aspirated he got blood just as though he had gone into a vein. Dr. Dwyer had reported that the blood was negative in all respects.

DR. HARRIS said that they had proceeded with the diagnosis to the extent of feeling that the tumor was largely angiomatous. The question of malignancy had not been decided upon. The fact that manual palpation gave fluctuation; and the history of the case, made him feel that it was not a case of broken down cyst; and that he was satisfied that what was found had been there for a considerable length of time, only that it was rapidly and continually growing larger. He promised to present the case again at the December meeting.

Hemi-Laryngectomy for Carcinoma of Larynx. DR. JOHN MCCOY.

The patient was first seen in April, 1917. He stated that he had begun to have hoarseness in October, 1916, and shortly after that pain on swallowing.

On examination, a papillomatous looking mass was seen on the left vocal cord and just below it. A section was removed for examination, and the pathologist reported that the condition was suspicious of malignancy. With that in view, it was decided to watch the case closely. In May the growth was removed, and the case was followed closely through May and June, during which period the growth remained quiescent. The patient returned in October, and it was then found that the growth had spread and infiltrated the arytenoid on that side. He was admitted to the hospital on October 25. A preliminary tracheotomy was performed, and a section removed from the growth. Examination proved it to be carcinoma, and on November 1 a hemi-laryngectomy was performed, completely removing the left half of the thyroid and cricoid cartilages. The wound was closed, and the patient made a good convalescence, with the exception that he developed an infection of the skin wound, which took three weeks to granulate.

In this case the three points were observed which are essential in the management of such conditions in order to secure a good result and normal convalescence: A preliminary tracheotomy a week before the operation; a thorough cleansing of the teeth and mouth; and rectal anesthesia. In this case, however, it was evident that the mouth was not as well cleaned as it should have been, for the skin infection was very suggestive of mouth infection. Nevertheless, the man was up on the third day and left the hospital three weeks after operation.

It is up to the laryngologist to take care of such cases. The growth was spreading and infiltrating the arytenoid, and it was possible to tell the patient what the condition was and to do what was necessary; whereas, if he had been sent to some one else he would probably have drifted away. He has now a good voice, can talk pretty well, and is a useful member of society.

DISCUSSION.

DR. HARRIS asked if there were any enlarged glands in the neck that required removal.

DR. COFFIN asked if he was correct in understanding that the patient was up and about in three days and upon receiving an answer in the affirmative he congratulated Dr. McCoy on the excellent result obtained. He then asked how the patient had been fed.

DR. MCCOY said that he had cut off half the larynx to the level of the esophagus and fed the man by a tube through the nose passing down through the pharynx and resting in the esophagus. This was continued for five days.

DR. YANKAUER asked if the tube was allowed to remain in place all the time.

DR. MCCOY replied in the affirmative.

DR. CARTER asked if any measures were taken to prevent contraction of the laryngeal opening? Was there any tendency to the formation of a stricture? He had examined the case and certainly both the Doctor and the patient were to be congratulated on the wonderful results attained.

DR. MACKENTY said that he was very glad to see a case of hemilaryngectomy, for the operation has been more or less discredited by laryngologists who claim that it has no place. He claimed that it has, but not a very large one. It was very gratifying to see such a good result. It would be interesting to know if Dr. McCoy had any trouble with the new wall which he formed. Was there any granulation tissue during the healing? Was any effort made to cover the new wall with flaps from any part of the throat in that region?

DR. MCCOY, in closing the discussion, said that the inquiry in regard to the glands brought up the important question of why laryngologists

should do these operations. This was a doubtful case: was it benign or was it malignant? A section was removed, but the pathologist could not determine the question. It was watched, and changed from an intrinsic growth and began to show evidence of becoming an extrinsic growth, with a much greater tendency to involve the glands; so it was decided to operate and probably thereby prevent the development of glands. That was the point he was anxious to emphasize. The laryngologist is the man to see and watch these border line cases, and the minute they show a tendency to infiltrate they should be operated upon, thereby preventing infection of the glands. No tendency to contraction had been noted. Nothing had been done to prevent stricture or granulations. That side of the larynx was formed practically by the sterno-hyoid, sterno-thyroid and thyro-hyoid muscles; the inside of the larynx on that side was covered with an exudate.

Replying to Dr. Yankauer he said that the tube was kept in place for five days.

Cyst of the Naso-Pharynx. Dr. Frank Henry Knight.

This case was seen in Dr. MacKenty's service at the Manhattan Eye, Ear, and Throat Hospital. He gave a negative history; his parents, four sisters, and two brothers are all living and well, and there have been no deaths in the family. He has never been ill since he was six years old, when he had scarlet fever. He is a mechanic, 26 years of age, born in this country.

On October 18 he came to the clinic complaining of a sense of fullness in the throat and a tickling sensation which caused him to cough, especially in the morning. There was no history of any particular post-nasal discharge. On looking into the oro-pharynx nothing was noted excepting a congested throat, probably due to smoking. On making a posterior-rhinoscopic examination, however, a tumor mass the size of a large walnut came into view. It was grayish in color, and at first was thought to be a polypus, but on testing further it was found to be attached to the posterior pharyngeal wall with a definite outline, one to one and a quarter inches in diameter. The mucous membrane was smooth, and there were two veins on the anterior surface, running down each side of the median line.

On introducing the pharyngoscope into the left nerve and passing it backward, another tumor mass came into view which seemed to originate in the pharynx. The Eustachian tube and Eustachian eminence of that side were not visible.

The case was interesting because of the two distinct tumors and because of its rarity. The one below was grayish in color and entirely distinct from the one above, which was covered by mucous membrane and similar in color to the other.

DISCUSSION.

Dr. MacKenty said it was quite possible that it was a congenital cyst, possibly from a branchial cleft. He had seen but one other case of this kind, even with the large clinical experience at the Manhattan Hospital.

Dr. Voislawsky asked if any attempt had been made to remove the boy's adenoids.

Dr. Carter said that several years ago he had a similar case, and the conclusion reached was that it was an agglutination of the adjacent sides of the adenoid tissue, and that the cyst formed in the enclosed tissue in this agglutinated area.

Dr. Yankauer said he had seen a number of abscess cavities formed by the agglutination of the adenoid tufts in this region—probably twenty—but he had never seen a cyst like this.

Dr. Harris said that some years ago he had reported a cyst like this before the American Laryngological Association. In that instance, the growth was examined microscopically, and the tissue determined the nature of its origin. At the time he looked up the literature and found that it was exceedingly rare. The patient was a woman of about 45,

and the growth was easily removed. It was interesting from the clinical standpoint, for there were very few cases on record.

DR. KNIGHT thanked the members of the Section for their discussion of the case. He thought that the remarks that had been made about its being a branchial cyst were probably correct, but he had hesitated to make that diagnosis until it had been examined microscopically. He had read the account of Dr. Harris' case, and also another of a patient who had several abscess cavities. Dr. Voislawsky had asked if the adenoids had been removed, but he could not answer that question. The boy said that he had never been sick in his life. Dr. Carter had spoken of the agglutinated area. Dr. Jonathan Wright has spoken of these cases as due to the elevation of the mucous membrane where it grew together and formed sinuses or cysts.

The main cyst seemed so large that it could not well be included in that class of formation, and accordingly he made the tentative diagnosis of pharyngeal cyst.

Auto-Esophagoscopy by a Patient. DR. HUBERT ARROWSMITH.

The case was one of more clinical interest than practical importance. The man was a sword-swallower by profession, and came to the hospital a week ago stating that he thought he had a nickle in his throat and had taken a sword to push it down, but could not. Dr. Arrowsmith said that the man objected to his method of performing esophagoscopy, and asked permission to put the tube in himself, which he did easily.

Gottstein has described a sword-swallower's esophagus.

The man was quite histrionic and took a great deal of pleasure in displaying his abilities. The reason he did not like Dr. Arrowsmith's method of passing the esophagascope was that the Doctor hurt his mouth. He makes his own swords, which are blunt from the springs of baby carriages and has swallowed six of them at a time. He would also swallow watches, as well as other articles of various kinds.

Dr. Arrowsmith said that the man had come to him complaining of a slight pain in his chest which troubled him. After some questioning, he said that a man had stabbed him in the right side, and then thrown him down stairs; upon which Dr. Arrowsmith told him that would probably account for the pain.

The esophagus was dilated, and the mucus membrane thickened and of coarser texture than normally seen.

DISCUSSION.

DR. EMIL MAYER said that it was a most interesting and instructive exhibit. Two points especially impressed him. First, the angle at which the man had tipped his head. He tilted it back, quite a good deal further than is usually employed by Laryngologists in the introduction of the esophageal tube. Second, after the tube was introduced about ten inches it made a decided jump, which would be about on the line of the cricoid cartilage, and then it slid in easily.

To the man it was merely child's play, but it formed a most interesting exhibit, demonstrating esophagoscopy as it should be done.

DR LYNAH said that it was a most remarkable exhibition and there was no doubt that the large swords the man was able to pass made the esophagoscopy easy. He had noted that the man carried out some of the suggestions made by Dr. Jackson. Another thing he did which Dr. Jackson has called attention to very often, the duration. The actual time was two or three minutes before he removed it.

DR. YANKAUER said that he had a patient who for a long time had been having the esophagoscope passed for cardiospasm. He does it in about

two seconds. There was little doubt but that this patient could pass the esophagoscope himself, and Dr. Yankauer said that he intended to have him try it.

Embryonic Carcinoma. DR. MACKENTY.
(To be published in a subsequent issue of THE LARYNGOSCOPE.)

Laryngectomy with Excision of the First Portion of the Anterior Wall of the Esophagus. DR. JOHN MCCOY.

The patient was a man 48 years of age, a native of Italy and a waiter by occupation. He gave a history that for the past five or six months he had experienced difficulty in swallowing, with special discomfort on the left side of the larynx; his voice had been affected and he had excessive secretion from the pharynx and larynx. Examination showed infiltration of the right arytenoid and interarytenoid space with ulceration on the esophageal face of the right arytenoid. Wassermann test was negative.

On August 8 a tracheotomy was performed under cocaine anesthesia and at the same time a portion of the growth was removed, which proved to be carcinoma. August 15 a total laryngectomy was performed. About one inch down in the esophagus a patch was found half an inch in diameter of typical carcinoma. The anterior portion of the esophagus was excised for about an inch in length. The wound was closed in the usual manner, the upper part being left open, and a tube was passed through the nose into the esophagus and allowed to remain for feeding.

In the after treatment, the wound was packed daily, the patient being fed through the catheter in the nose. He convalesced nicely and was up in four or five days, walking about, and had practically no reaction. At the end of a week, ten Mg. of radium was introduced into the esophageal opening; this was repeated a week later, and again a week after that. The patient was on the roof of the hospital on the sixth day. Three weeks after the operation, while being fed through the catheter, which was attached to his cheek, the catheter was loosened and he took hold of it, and while the nurse was putting in the tube he let go of the catheter, and swallowed it. This did not seem to disturb him, however, and he progressed as if nothing had happened. After several days, an x-ray picture showed the catheter in the stomach. Another picture, taken a week later, showed the catheter still in the stomach. It was then decided to give him an anesthetic and pass the esophagoscope and search for the catheter, after which the skin of the neck was to be sutured to the esophagus. He had entirely healed, and one could look into his esophagus; it was intended to sew the side of the neck to the esophagus—the anterior half of which had been cut away—with the idea of later loosening flaps on either side and closing over with them.

This was done on September 13. Unfortunately, however, rectal anesthesia was not employed, but the anesthesia was administered through the tracheal opening in the neck and a considerable amount of ether had to be used to relax the stomach sphincter. The skin of the neck was then sutured to the esophagus. Twenty-four hours later the patient developed a pneumonia to which he succumbed after four days. He had been well for a month and by the mistake of giving him ether through the tracheal wound the pneumonia developed and the excellent results that had been obtained were destroyd.

DISCUSSION.

DR. MAYER said that he had been very much interested in the case reported by Dr. MacKenty. It recalled a case he had seen a number of years ago with the late Dr. Asch. It was that of a young boy and it involved practically all of the sinuses, the eyes were bulging and was a

most painful and distressing sight. The patient, fortunately for him, died very soon after the speaker had seen him and there had been no attempt at operation.

The speaker asked Dr. MacKenty if in view of the inevitably fatal outcome of the case if he would not in the future prefer to refrain from operating and use Radium only?

DR. FREUDENTHAL, referring to Dr. McCoy's case, said that Dr. McCoy stated that the catheter slipped down the esophagus and then could not be found. Presumably, he used the direct method. It was strange that we do not more frequently employ a method recommended years ago, which he had employed once with satisfactory results, i. e. the use of the x-ray in searching for the foreign body. The patient is put on the table and the tube under it, and then the examination is made with the fluoroscope either by the operator or by his assistant. This gives excellent guidance. It would seem that such a large instrument as a tube could be found easily by such a method, especially since it had been discovered before by the x-rays.

DR. YANKAUER said that rubber would probably not show at all, not even hard rubber.

DR. MACKENTY, replying to Dr. Mayer's inquiry as to whether or not he would follow the same procedure in another case, said that it was Dr. Janeway's opinion that one has to get pretty well down to the base of the growth to get good results with radium. With sarcoma it is different, but with carcinoma radium does not give a very good result. It is better to remove the growth and apply the radium immediately afterward. That proved to be so in this case. Dr. Janeway used a maximum dose. The effect of the radium on retarding the return of the growth was very remarkable.

Intubation Tube Removed From Right Lung After Two Years. DR. HUBERT ARROWSMITH.

A. F., a girl 4 years and three months old was brought to King's County Hospital in May, 1915, suffering from dyspnea. It was assumed by the House Staff that she had laryngeal diphtheria and she was immediately transferred to Kingston Avenue Hospital where she remained from May 25 to June 7, 1915. Diagnosis: "Laryngeal diphtheria, no visible exudate; aphonia following a surgical operation, with slight chronic laryngeal stenosis."

No definite previous history could be obtained, but we assume that the intubation was done prior to her admission to Kingston Avenue Hospital and that the tube had been inhaled before she came under observation there. This is a 2 year tube; too small for the patient. Subsequently a diagnosis of laryngeal papilloma was made in another Brooklyn Hospital. A tracheotomy was done; later a number of attempts failed to relieve the laryngeal stenosis.

She came into my service at King's County Hospital June 26 last. General condition bad, with a continuous and copious discharge of foul smelling pus around and through the tracheotomy. On physical examination but little air entered the right lung and everywhere there were coarse bubbling rales.

Radiography showed the tube lying with the head apparently in the right main bronchus and the long part of the tube in the lower lobe stem.

Direct laryngoscopy showed the larynx to be practically impervious. Lower bronchoscopy through the tracheotomy wound revealed an almost complete and resistant stenosis of the right main bronchus from which a large quantity of pus exuded. A small portion of the head of the tube could be distinguished through the swollen bronchus. Repeated attempts to grasp the tube by various forceps failed, as the infiltration was too great to allow the jaws to pass over the head of the tube.

The bronchscope was withdrawn, the light carrier passed into the bronchus and with Lynah's introducer-extractor, which was readily inserted in the tube opening, we were equally unsuccessful, the instrument always slipping out without budging the tube.

A week later a 9 mm. Brüning bronchoscope was passed, the bronchial walls dilated rather forcibly by a strong pair of alligator forceps, Lynch's knot tier inserted in the lumen of the intubation tube, a slight rotation back and forth loosened it and it was easily withdrawn.

The patient's general and local condition has steadily improved and a laryngostomy to relieve her laryngeal stenosis is planned for the near future.

Two Cases of Esophageal Diverticulum, One Double. DR. H. ARROWSMITH.

The first patient was a man aged 78 who for several years had suffered from gradually increasing difficulty in swallowing and regurgitation of food. He was esophagoscoped and the large diverticulum shown in the radiographs was found. It measured one and one-half inches in depth and two inches transversely.

Dr. George K. Meynen, of Jamaica, and Dr. Arrowsmith, assisted by Dr. Lynch, did a Jackson-Gaub operation.

Up to the seventh day, the history was uneventful, temperature not over 100° by rectum, the patient sat up from the third to the seventh day and was satisfactorily nourished by a pernasal tube which was left continuously in place. On the morning of the seventh day he had a severe chill and a red and painful swelling appeared in the supra-clavicular region of the side *opposite* to the side of operation. On palpation there was a distinct crackling and it was assumed that there was a gas bacillus infection. Death occurred on the eighth day. An esophagoscopy immediately after death showed the diverticular wound entirely healed. The neck wound was opened and there was practically no suppuration. The esophagus was removed through the operation wound and was lacerated in the manipulation. A culture was made, but nothing characteristic developed.

The second patient was a man 43 years of age, with a very similar history, who had *two* diverticula, one below the other. The speaker had not been able to find mention of double diverticula in literature. He purposed to operate on this patient also by the Jackson-Gaub method.

Foreign Body Removed from the Bronchus. DR. SIDNEY YANKAUER.

Dr. Yankauer said that he had two cases to report, both of unusual interest. The first was that of a child, two years old, referred by a physician with the statement that the child had been sent to him with a history of having had tonsilitis four weeks previously. Since the tonsilitis the child had had an inspiratory stridor, but the doctor stated that nothing had been said to him about the intubation.

The child was examined in the office with the direct laryngoscope, and a foreign body was found in the trachea, below the vocal cord. It had the shape and appearance of a disc of brass, and might have been a small coin. Dr. Yankauer said the condition seemed so simple that he could not resist the temptation to try to remove the foreign body, and he seized it with forceps, but he could not budge it up nor down. The child was sent to the hospital for a second attempt to be made, with the aid of suspension laryngoscopy. In the afternoon, he was informed that the child had become dyspneic and a tracheotomy had to be performed. The child was relieved, and an x-ray picture was taken to determine the size and shape of the foreign body. The next morning the radiographer reported that the plate failed to show any foreign body. Another direct laryngeal ex-

amination was made, but to his surprise, no foreign body was found. Accordingly he had other x-ray pictures taken, from the head to the foot, and the stools were watched, and the child examined most carefully. The next morning the foreign body had not been found in the stools. The child had been radiographed from the top of the head down, and no foreign body had been found, and it had not been coughed up, yet at the first examination a piece of brass which was perfectly distinct had been found. This had disappeared, and had thrown no shadow on the x-ray plate.

The child had had a tracheotomy performed, and it was a very small matter to do a lower bronchoscopy, and when the right bronchus was reached, the foreign body was found a little below the bifurcation. This was seized with the forceps and removed very easily, and the mystery was solved; all the examinations were right and all wrong, for the foreign body proved to be a piece of glass, the edge of which was gilded.

The second case was that of a child 3 years of age, admitted to the medical service with a statement that she had been suffering from pneumonia for two weeks. The child was observed for a week without any amelioration of the symptoms. There were all the usual signs of pneumonia on the right side, involving two-thirds of the chest. Aspiration was performed and pus withdrawn, so the surgeons were called in to open the chest. Then a radiograph was taken, but it did not correspond to the usual picture of empyema. The outer portion of the chest showed clear to the diaphragm. Accordingly, Dr. Lilienthal, who had had some similar experience, refused to open the chest and suggested bronchoscopy first.

At the bronchoscopic examination, the bronchus was found to be considerably swollen and there was some difficulty in passing the tube, but this was overcome and a gush of pus, fully an ounce, poured out of the end of the bronchoscope like water from a spigot. When this ceased, a foreign body was seen, which proved to be a piece of nut. This was removed, and a second piece also was taken out.

The next day the child's temperature came down, and it has been steadily improving since. Whether or not it would make a complete recovery, Dr. Yankauer was not prepared to say, for cases have been seen where vegetable substance had been in the lung and lung abscess developed later, in spite of their removal. This child has had pneumonia for two weeks, and while it may recover, it might have further trouble.

Both cases were examined without any anesthesia.

Button in Esophagus. DR. J. D. KERNAN.

The patient, a child of fifteen months, was admitted to Dr. Harris' service in August. The mother gave a history that the child could not swallow its accustomed solid food. It could take its bottle all right, but if it was given bread or other solid substance it would vomit. Two radiographs were taken, and both were reported negative for foreign body, but on account of the history of esophageal obstruction an esophagoscope was passed, and a foreign body was seen. Just as we were prepared to grasp it, it slipped down, but eventually it was recovered.

The two points of interest in the case were that so solid a substance as the button should have cast no shadow, though after the button had been found it was thought that a faint indication could be found on the plate. The second point concerns the difficulty in grasping the foreign body, after it was seen. Many experienced men have told of the difficulty they have had in releasing foreign bodies from the esophagus and of the ingenius methods which have finally succeeded. Dr. Kernan said that in this case he had had the experience of having the foreign body slip downward of itself and also in another where a large tooth plate passed downward in similar manner to be recovered afterward only with difficulty.

DISCUSSION.

DR. HARRIS asked if Dr. Arrowsmith had thought of the Mosher operation brought forward a year or more since, working by that method in connection with the second operation which he is about to perform.

In regard to the case reported by Dr. Kernan, Dr. Harris said that he had seen the case and had tried to remove the foreign body before Dr. Kernan was given charge of it, but had not been able to see it at all. The point which Dr. Jackson has so often made was demonstrated in this case. One should not attempt to do any foreign body work unless equipped with all the necessary instruments and prepared to do anything that may arise. When the first attempt was made to find this foreign body, they meet the usual experience of every large hospital: the equipment was faulty, and they did not have the proper forceps. Dr. Kernan had his own forceps, and succeeded. That point should be emphasized in all instances. We should keep our hands off unless we are provided with all the instruments required to meet any situation that may arise.

DR. MAYER said that Dr. Chevalier Jackson has a large screen on which he has pictured some of the many foreign bodies which he has removed, one side is labeled "hardware" and the other side "toys." It would be interesting to know how he would designate the piece of glass shown by Dr. Yankauer. It was a remarkable case and Dr. Yankauer might have been forgiven if he had not found the glass, for it was a transparent object, singularly difficult to see and the x-ray gave no help. It spoke highly of the Dr.'s keenness of vision as well as his accuracy of work.

The speaker would like to know whether there was not much difficulty in removing vegetable substances as shown in his second case. Was there not much danger in removing them with a forceps when they were softened, crushing them into a pulp? Did he ever try washing them out?

DR. ARROWSMITH said that the great objection to the Mosher operation referred to by Dr. Harris, though he had never tried it, seemed to lie in the difficulty of convincing oneself, that the diverticulum and the esophagus have a party wall; when absent if you attempt to slit the wall between you get into the mediastinum, and that would be quickly fatal. Dr. Arrowsmith said he did not see how one could determine beforehand whether there was a party wall.

DR. LYNAH said that he had had the pleasure of seeing these cases with Dr. Arrowsmith, especially the intubation case, and had thought at first that Dr. Arrowsmith had put one over on them, but after the tube was removed he proved an alibi. The child had been in some Brooklyn hospital where the tube had been inserted, and was to go back for further treatment. The mother said that some doctor told her that a tube had been put in, and in endeavoring to take it out the tube had been pushed down. That is not an infrequent occurrence. Just before Dr. Arrowsmith showed his case, he had seen a similar one in another hospital. Drs. Grove and Dixon at the Willard Parker Hospital had each removed one or two. The accident was not infrequent.

It was a great disappointment that Dr. Lynah should lose his patient after he was doing so well.

The point that Dr. Arrowsmith made was very important—that after he had located the diverticular orifice and was passing the bougie he used a larger esophagoscope with the bougie. That gave the surgeon ample lumen in which to operate. That was the first time he had heard of such a thing being done.

Dr. Yankauer was to be congratulated, as usual, on his brilliant results in foreign body cases, especially in the one with the glass. Dr. Jackson would probably put that in his crystal palace.

As for the nut kernel, vegetable substances are always irritating and are not always completely removed, and frequently result in abscess of the lungs.

Referring to Dr. Kernan's case, Dr. Lynah said that he had recently seen a case in which the foreign body was a pearl button which had been in place for a week or ten days, and it showed a very faint shadow in the radiograph. When grasped with the forceps, a little piece came off; the rest had been dissolved by the hydrochloric acid of the stomach. On the strength of that, Dr. Grove tried a similar button in hydrochloric acid; at the end of a week or ten days it was completely dissolved.

DR. HELLER said the reason the button dissolved in the esophagus was that it is formed of casein. The so-called mother-of-pearl ordinarily used in cheap buttons and knife handles does not contain a particle of pearl. It is made up largely of casein compressed by a special process. The action of the acid gastric juice is exactly that of a tough milk curd.

DR. YANKAUER asked whether the Doctor had the dilating forceps of Bruening made for just such work. It had two blades that could be introduced and then opened, and very sharp teeth.

Referring to his own case, Dr. Yankauer said that neither gave any history whatever of inhaling any foreign body. In the case of the child with the glass, it was later found that when the child had tonsilitis they tried to give it a dose of castor-oil.

DR. MAYER had asked about removing vegetable substances. The difficulties with these vary a great deal, according to their constitution and the length of time they have been in place. Some of them become pasty and it is impossible to remove them. When they have been masticated, it is very difficult to remove them. In one instance a peanut had been inhaled. He removed as much as possible, and then inserted a spoon and removed more, and still the child developed an abscess. The best precaution is to be sure that one has an instrument that opens and closes lightly, so that one can gauge the pressure of the foreign body, and then, if one has a delicate sense of touch, he can tell just how much pressure to exert and can remove the object with the least possible breaking.

PHILADELPHIA LARYNGOLOGICAL SOCIETY

Meeting in Thomson Hall, College of Physicians, January 8, 1918.

Demonstration of the Internal Anatomy of the Face and Associated Parts (Illustrated). DR. MATHEW H. CRYER (by invitation).

Dr. Cryer elaborately and clearly demonstrated the relation of the sinuses to each other and to the teeth and advocated the dental route for drainage of the antrum. Dr. Cryer displaced a large assortment of abnormal specimens of sinuses, large as well as small maxillary and frontal sinuses.

Dental Pathology with Special Reference to Diseases of the Nose, Throat and Ear. DR. WM. STIRLING HEWITT (by invitation).

Dr. Hewitt stated that the pathology was no different from pathology of any other part of the body, but emphasized thorough study of the teeth when diagnosing conditions of the nose and throat. Also, that the X-ray is too often neglected.

DR. HERMAN B. COHEN, *Reporter.*